



**THE UNIVERSITY OF CHICAGO
THE PRITZKER SCHOOL OF MEDICINE**

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Attention:

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Re: Rodriguez vs Union Pacific Railroad Company

Dear Mr. Brian Winegar:

Please find a report regarding my opinions on this case below.

1. I am a physician, duly licensed to practice medicine in the State of Illinois. I am Board Certified in Medical Oncology and practice in the fields of Medical Oncology with a subspecialty in Gastrointestinal Oncology, including patients with colorectal cancers from both clinical and research perspectives. I have been involved in these patients' care including various therapies such as in the perioperative and metastatic settings.

At the University of Chicago, I am the Director of Gastrointestinal (GI) Medical Oncology Program. This entails overseeing a clinical program entailing 6 GI Medical Oncology Faculty members, 3 Advanced Nurse Practitioners, 5 Nurse Navigators, and a Pharmacist, as it pertains to operations of the clinical and research programs within GI Medical Oncology. Annually, we have a census of > 1600 GI cancer patients, of which >350 are new patient and new consultation visits. In addition, I oversee and run the research program entailing 4 clinical trial coordinators, 4 data managers, regulatory personnel, and biobank personnel. The GI research program has more than 30 investigational clinical trials open at the moment. The extends to 3 community satellite centers of the University of Chicago where our studies are available there. As the Director of Interdisciplinary GI Oncology and Assistant Director of Translational Research, this research focus extends to the other oncologic disciplines of GI Surgical Oncology, Radiation Oncology, as well as Anatomical and Molecular Pathology, where I oversee and facilitate cross-discipline collaboration and research.

I have published numerous publications focusing on the management of GI cancers, as well as biologic mechanisms and novel therapeutics of these diseases. I have presented these topics and my research findings internationally at medical conferences and by invitation to academic centers. I have obtained NIH research funding, foundation awards, collaboration with biotech and pharmaceutical companies, and philanthropy to support my work. A primary research focus of mine is on the biological understanding and treatment of gastroesophageal (esophagus, gastroesophageal junction, and stomach) cancers, by studying the normal and oncologic components and molecular pathways of gastrointestinal cells. My research agenda has an overarching goal to validate and improve personalized treatment, immunotherapy, and precision medicine for gastroesophageal cancer and other GI cancers, with findings often relevant to all cancers. A major component of my research is on the quantification of tumor genetic molecular heterogeneity both between individuals with gastroesophageal cancer, but importantly also within a given individual within one tumor site, and from one tumor site to another, and how this impacts personalized targeted therapeutic approaches. To overcome many biological hurdles of the disease that has led to failed therapeutic approaches in the past 1-2 decades, I have designed and executed novel clinical trials to implement treatment strategies based on these laboratory and clinical discoveries.

I serve as a mentor to medical trainees including medical students, internal medicine and surgical residents, as well as medical oncology and surgical oncology fellowship trainees. Most teaching is part of clinical training during clinical care of patients in the inpatient and outpatient setting. I also teach formal didactic lectures to these trainees on the topics of management of various GI cancers. I also teach didactic lectures to first and third year Graduate Students in Cancer Biology regarding the biologic underpinnings of GI cancers and therapeutic strategies.

I serve as associate editor for the *Journal of American Medical Association Network Open (JAMA Netw Open)*, and I am also on the editorial boards of the *Journal of Clinical Oncology Precision Oncology (J Clin Oncol PO)*, *Cancer*, and *Cancers*. As associate editor for the Oncology section of *JAMA Netw Open*, I review manuscript submissions pertaining all cancers and from all disciplines (medical, surgical, and radiation oncology) to the journal and determine which manuscripts will be sent for external peer review versus those that would be rejected without review. I then review those manuscripts and external peer reviewer comments and provide a final decision as to whether to reject or accept them paper for publication. As associate editor of *JAMA Netw Open* and member of the editorial boards of *J Clin Oncol PO*, *Cancer*, and *Cancers*, I attend regular board meetings to discuss papers and general operations of the journals. I also serve as an ad hoc reviewer for numerous journals to serve as an external peer reviewer to provide comments and recommendations on acceptance of manuscripts, pertaining to GI cancers, submitted for publication.

I am a member of many medical societies and groups, including the American Society of Clinical Oncology (ASCO), European Society of Medical Oncology (ESMO), the American Association of Cancer Research (AACR). I have participated in consensus guidelines for the treatment of GI cancers for ASCO and other Consortia.

2. I was asked to provide an opinion on exposure of asbestos and its contribution towards causation of Mr. Rodolfo's colorectal cancer and untimely death.

As such, I have reviewed information and records regarding the case of Rodolfo (Rudy) Rodriguez, including the medical records from Big Bend Regional Medical Center and Midland Memorial Hospital regarding his diagnosed *KRAS* mutated colorectal (recto-sigmoid) cancer. I have reviewed the death certificate of Rodolfo Rodriguez having date of death 10/10/2017 and an

indication due to metastatic colon cancer. I have reviewed the letter of Dr. Courtney Crim from 6/11/2020. I have reviewed the depositions of Rito Ortega, Daniel Rodriguez, Diana Rodriguez, Jose Rodriguez, and Rosamaria Gomez Rodriguez. I also reviewed the written report of Mr. Richard Miller. Additionally, I performed a literature review to provide my written report to opine on these topics.

3. Introduction to Cancer:

Cancer is the abnormal and uncontrolled growth of cells in the body.¹ Cancer cells are a distorted version of a normal cell – it is well-established that cancer arises from alteration of one or more cancer-related genes due to change of the DNA sequence and/or changes in the amount of DNA (amplification/deletion), or the expression of the gene itself (through epigenetic changes). Cancer-related genes can be one of two main categories: tumor suppressor genes or oncogenes. Tumor suppressor genes are the ‘brakes’ in the system, and signal for cells to stop dividing/growing and if there is severe damage to the cell, the signal for it to die (apoptosis or senescence).² Oncogenes are the ‘gas pedal’ of the system, signaling for cells to divide and increase in number, grow in size and also some oncogenes signal the cell to migrate to other areas in the body.¹ Normally, tumor suppressors and oncogenes signal in concert and in equilibrium with each other to maintain a balance, called homeostasis. If there is a wound, nearby cells will be signaled to divide, grow, and migrate to the wound to heal it, but when healed, the cells will return to steady-state. Cancer cells signal to grow inappropriately, due to altered DNA, and behave like a wound that never stops healing. Cancer cells continue to grow inappropriately and the ratio of cell growth to cell death increases, and therefore often cancer masses will form, referred to as ‘tumors’. However, some tumors grow as single invasive cells in the absence of classic tumor formation, called diffuse type tumors, such as signet ring gastric cancers.

Although different cancers from different sites and tissues of the body have different sets of altered genes causing the cancer, ultimately, all cancers are caused by alterations in the DNA.³ These alterations of the DNA within cancer-related genes may be inherited, induced by environmental factors, from random DNA replication errors, or a combination of these factors. A carcinogenesis model has been described for various cancers specifying common genes altered and the sequence in which this occurs over a period of several years. It has been estimated that at least half of the genetic changes occur in precursor cancer cells prior to formation of any tumor mass.

Inherited pathogenic alterations, called germline alterations, can be from a single highly penetrant gene (eg. a tumor suppressor like the APC gene in colorectal cancer),^{4,5} or they can be weaker penetrance and also can be multifactorial (multiple causative genes, but each contributing to the develop of cancer to a small degree) and more difficult to discern. A germline event(s) is present prior to the formation of the zygote (the one cell made up of DNA that is half from one parent and half from another parent, also known as a fertilized egg) in the DNA of one (or both) parents. It is estimated that inherited genetic factors are causative or contributory to approximately 5-15% of cancers, depending on the cancer type. Inheriting an altered pathogenic gene usually leads to the onset of a cancer at a younger age, due to the carcinogenesis model shifting earlier in time (ie the cancer development gets a head start right from development).

On the other hand, somatic alterations are those that occur after conception of a zygote, through gestational development, and then after birth and through an individual’s lifetime. Somatic genetic alterations can occur from environmental exposures and/or from random DNA replication errors, also referred to as stochastic effects associated with the lifetime number of stem cell divisions within each tissue.

Environmental factors that contribute to the cause of cancer have been described, and can be specific to certain cancer types.⁶ Environmental factors include aspects of lifestyle, economic, and behavioral exposures. Chronic inflammation, through various etiologies including infection or other agents, has been associated with carcinogenesis. Poor diet,⁷⁻⁹ inactivity, and obesity¹⁰ have each been associated with carcinogenesis. Some specific foods are linked to specific cancers. Regardless, any factor that may alter DNA sequence and contribute to carcinogenesis and the ultimate development of cancer can be referred to as a carcinogen. Carcinogens (also referred to as mutagens) are substances or agents that promote DNA changes leading to cancer. Tobacco smoke, for example, is a common and well-known to contain over fifty carcinogens, including nitrosamines and hydrocarbons. In addition to chemicals, radiation and radioisotopes are known carcinogens. Infections with certain viruses, bacteria, and worms are also known carcinogens. Environmental agents such as asbestos are known as carcinogens. Endogenous or exogenous hormones drive cell growth and are known carcinogens. The International Agency for Research on Cancer (IARC) has listed groups of agents into categories (Group 1, 2A, 2B, 3, 4) based on the strength of available evidence supporting it as a carcinogen as follows¹¹:

Group 1: the agent (mixture) is definitely carcinogenic to humans. The exposure circumstance entails exposures that are carcinogenic to humans.

Group 2A: the agent (mixture) is probably (product more likely to be) carcinogenic to humans. The exposure circumstance entails exposures that are probably carcinogenic to humans.

Group 2B: the agent (mixture) is possibly (chance of product being) carcinogenic to humans. The exposure circumstance entails exposures that are possibly carcinogenic to humans.

Group 3: the agent (mixture or exposure circumstance) is not classifiable as to its carcinogenicity to humans.

Group 4: the agent (mixture) is probably not carcinogenic to humans.

Cancers are classified by the cell type of origin.¹² The most common cancers are carcinomas, those derived from epithelial cells, such as the mucosal lining of the GI tract. Sarcomas are another group of cancers that arise from the connective tissue like muscle, bone, and cartilage with precursor cells called mesenchymal cells. Malignant hematopoietic cells (leukemia and lymphoma) arise from blood-forming cells in the bone marrow and or lymph tissue in the body. Other less common cell types of origin are germ cell tumors (derived from pluripotent stem cells and in tissues such as testicle and ovary), or blastomas (cancers derived from immature precursors cells or embryonic tissue).

4. Introduction to Colorectal Cancer:

Colorectal cancers are cancers that arise in the large colon, which is comprised of the Cecum/appendix, ascending colon, transverse colon, descending colon, sigmoid colon, and rectum. The cancer precursor cells arise from the epithelial cells (the mucosal inner lining of the colon), and are carcinomas that have mucus gland differentiation, and are thus 'adenocarcinomas'. Annually, approximately 147,950 new cases of large bowel cancer are diagnosed, 104,610 of which are colon cancer, and the remainder are rectal cancer.¹³

Colon cancer etiology has been associated with a number of contributing factors,¹⁴ including genetic syndromes,^{4,5} high fat and red meat diet,¹⁵⁻¹⁷ obesity,^{10,18} sedentary lifestyle,^{19,20} diabetes,²¹ radiation,²² inflammatory bowel disease,²³ asbestosis (see below),^{24,25} tobacco and alcohol (see below)²⁶. It is common to have more than one associated risk factor, and it is likely that having many factors will heighten the risk of developing colorectal cancer.

Colon cancers start at level of an individual cell within the mucosa (the most superficial layer in the colon that serves as the inner layer of the ‘tube’) that acquires genetic alteration.²⁷ The initial pathology is the formation, usually, of a polyp.²⁸ A polyp is mass of hyperplastic cells that forms a pedunculated polyp into the colon lumen. Over time, accumulation of more genetic alterations in tumor suppressor and oncogenes within cells in the polyp lead to a ‘transformed’ and invasive component of the polyp which is malignant.²⁹ Over time, the invasive cancer cell mass proliferates and can become large in its site of origin and also start to invade into deeper layers below the superficial layer of the mucosa. Over more time, the cancer can acquire more genetic mutations, and also travel through the lymphatic system and/or the blood to spread to distant sites in the body. This is called a ‘carcinogenesis model’, and that model of colorectal cancer and aberrant gene acquisition and cancer spread over time is well-established.³ Staging of the cancer at the time of diagnosis determines the patients’ prognosis and treatment course.^{30,31}

Colon cancers are staged from I-IV:

Stage I – the cancer invades deeper into the submucosa or the next layer, the muscularis propria.

Stage II – invade from the inner surface of the colonic mucosa to the

Stage III – metastatic to regional lymph nodes.

Stage IV – metastatic to sites outside of the original site of origin and regional lymph nodes

Surgery is recommended for patients with stages I-III and a subset of ‘oligometastatic’ stage IV (few distant metastatic sites) with curative intent. However, the recurrence risk of stages II-IV who have undergone curative intent surgery is relatively high, and chemotherapy has shown to decrease the risk of recurrence.³² However, stage IV non-oligometastatic is currently incurable, and treatment is considered palliative; patients ultimately will die from their cancer with a median survival of 24-36 months from diagnosis of stage IV disease.³¹

5. Exposure:

I reviewed the depositions of Rito Ortega, Daniel Rodriguez, Diana Rodriguez, Jose Rodriguez, and Rosamaria Gomez Rodriguez. The deposition of Rito Ortega, a coworker of Mr. Rodolfo Rodriguez while at Southern Pacific Railroad Company, indicated the use of ‘magic rope’ used on cold days and almost every day during the winter months where it was soaked in diesel, put on the rails, and lit to heat the rails when making repairs. Jose Rorigeuz also worked as a welder helper while Mr. Rodolfo Rodriguez was a trackman and truckdriver, and they worked together every day during his time at the railroad. Jose Rodriguez remembers ‘asbestos powder from bags in the warehouse’ that was used to ‘mix with water and apply to bond wires on tracks’. He also recalls the use of ‘magic rope.’

I also reviewed the expert report of Mr. Richard Miller, who concluded that Mr. Rodolfo Rodriguez was ‘more likely than not exposed to asbestos fibers and carcinogenic diesel combustion products’ and ‘these exposure events would have substantially increased Mr. Rodriguez’ risk for cancer’.

“Thus, in summary, it is my professional opinion that Mr. Rudy Rodriguez was, more likely than not, exposed to asbestos fibers and carcinogenic diesel combustion products from the use of the so-called “magic rope” in his daily work repairing rails near the Sierra Blanca and Fort Hancock, TX sites. Additionally, the testimony indicates he often rode a “work train” to the site, therefore, during these time periods, Rudy Rodriguez was more likely than not exposed to the carcinogen components of diesel exhaust as well as the combustion products of the diesel fuel associated with

the burning of “magic rope.” All of these exposure events would have substantially increased Mr. Rodriguez’ risk for cancer.”

Finally, I reviewed the letter of Dr. Courtney Crim from 6/11/2020, where it is clear that his opinion is that Mr. Rodriguez had parenchymal and pleural abnormalities supportive of asbestos-related disease.

“This letter summarizes the NIOSH B-read for Rodolfo Rodriguez dated 10/11/2016. The chest film was provided on a drive. The film quality was grade 2 as there was overlay of the scapulae. Parenchymal abnormalities of s/t size and shape were noted in the lower zones bilaterally of profusion 1/0. In the setting of appropriate occupational exposure, this finding is supportive of asbestos. There were pleural abnormalities observed, supportive of asbestos-related disease in the form of *en face* plaques bilaterally. The plaques showed no evidence of calcification and extended more than one-half the length of the chest wall. Other abnormalities noted included an ill-defined hemidiaphragm. Sincerely, Courtney Crim, M.D.”

Based on the review of the above materials, and in addition my own qualitative review and a review of the literature, it is my opinion is that Mr. Rodolfo Rodriguez had ample exposure to asbestos during his time at Southern Pacific Railroad Company.

6. **Asbestos:** Asbestos is the commercial name for a group of hydrated magnesium silicate fibrous minerals²⁴. Asbestos occurs naturally in soil and rock as long fibers. There are two major types: serpentine and amphibole. All asbestos fiber types are carcinogenic and pose a threat to human health. About 95 percent of the asbestos produced and used commercially worldwide is chrysotile, which is a serpentine fiber.³³ Asbestos has been valued for its resistance to heat and combustion. It is used in cement, ceiling and pool tiles, automobile brake linings, manufacture of train and locomotive components, and in shipbuilding. It is well known that workers (and family through indirect contact) with asbestos exposure are at significant risk for the development of both non-malignant and malignant pulmonary disease, particularly mesothelioma and lung cancer. The lifetime risk of developing mesothelioma among asbestos workers is thought to be as high as 10 percent, with classic latency of approximately three decades from time of initial exposure.³⁴

When asbestos fibers in the air are inhaled, they can stick to mucus in the throat, trachea (windpipe), or bronchi (large breathing tubes of the lungs) and might be cleared by being coughed up or swallowed. But some fibers reach the ends of the small airways in the lungs or penetrate into the outer lining of the lung and chest wall (known as the *pleura*). These fibers can irritate the cells in the lung or pleura and eventually cause lung cancer or mesothelioma. However, asbestos fibers can also be swallowed.³⁵ This can happen when people consume contaminated food or liquids (such as water that flows through asbestos cement pipes). And, it can also occur when people cough up asbestos they have inhaled, and then swallow their saliva. As a consequence, asbestos workers have had a demonstrated increased risk of non-mesothelioma gastrointestinal malignancies.³⁶⁻³⁹ In the earliest report of such an association, Selikoff et al reported that, “Of 632 insulation workers, who entered the trade before 1943 and were traced through 1962, forty-five died of cancer of the lung or pleura, whereas only 6.6 such deaths were expected. Three of the pleural tumors were mesotheliomas; there was also one peritoneal mesothelioma. Four mesotheliomas in a total of 255 deaths is an exceedingly high incidence for such a rare tumor. **In addition, an unexpectedly large number of men died of cancer of the stomach, colon, or rectum (29 compared with 9.4 expected).** Other cancers were not increased; 20.5 were expected, 21 occurred. Twelve men died of asbestosis.”³⁸

IARC classifies asbestos as a Group 1 carcinogen (“the agent (mixture) is definitely carcinogenic to humans. The exposure circumstance entails exposures that are carcinogenic to humans.”) The National Toxicology Program (NTP) is formed from parts of several different US government agencies, including the National Institutes of Health (NIH), the Centers for Disease Control and Prevention (CDC), and the Food and Drug Administration (FDA). The NTP has classified asbestos as “known to be a human carcinogen”, as does the US Environmental Protection Agency (EPA).³⁷

7. Asbestos and Colorectal Cancer:

In 2009, the International Agency for Research on Cancer (IARC) evaluated asbestos and concluded, “There is *sufficient evidence* in humans for the carcinogenicity of all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite). Asbestos causes mesothelioma and cancer of the lung, larynx, and ovary.” “Also positive associations have been observed between exposure to all forms of asbestos and cancer of the pharynx, stomach, and colorectum.” “For cancer of the colorectum, the Working Group was evenly divided as to whether the evidence was strong enough to warrant classification as *sufficient*.” IARC stated, “All forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite) are *carcinogenic to humans* (Group 1)” (IARC 2012). I believe there is ample evidence that asbestos causes colon cancer. Much of this evidence has come out since 2012, and my findings are discussed below.

Preclinical Mechanistic Animal Studies:

A number of preclinical studies have been conducted supporting a causative effect of asbestos on colorectal cancer carcinogenesis. One showed that ingested chrysotile asbestos can alter regulation of DNA synthesis in the gastrointestinal mucosa in rats.^{40,41} Another study evaluated the long-term effects of ingestion of asbestos on the colon in rats, and observed and concluded that i) Chrysotile fibers were seen by electron microscopy in six of ten colon specimens of rats fed and asbestos diet; ii) evidence of increased probability of asbestos-fed animals to develop colon lesions in general; iii) evidence for a cell regulator defect (lowered cAMP levels) in colon tissues of animals fed asbestos; and iv) evidence for asbestos fiber penetration of the colonic mucosa (electron microscopy studies) suggest that ingested asbestos is not inert in the colon.⁴² In another study, the mucosal lining of the colon underwent changes consistent with a mineral-induced cytotoxicity after ingestion of chrysotile asbestos.⁴³ Asbestos fibers interact with mucosal cells of the GI tract causing damage and death of superficial cells and they also penetrate intestinal mucosa both in vivo and in vitro,⁴⁴ leading investigators to conclude that asbestos acts like a classical tumor promoter.⁴⁵ Cell proliferation caused by asbestos leads to tumor development and promotion, and the ability of chrysotile to stimulate cell proliferation, using a number of biomarkers, has been demonstrated both in vitro and after inhalation by rats.^{36,46} In one study, crocidolite asbestos induced abnormal crypt foci in the colon of rats in two independent experiments ($P = 0.02$ and $P < 0.01$ compared to controls given water), and chrysotile asbestos also induced abnormal crypt foci.⁴⁶ There are some studies that have been conducted that have not demonstrated casual effect of asbestos on carcinogenesis, for example Bolton et al, although the numbers of animals evaluated in that study were low.⁴⁷ Overall and taken together, it is my opinion that, collectively, these such studies provide a mechanistic carcinogenesis effect of asbestos exposure towards colorectal cancer development.

Human Tissue Studies:

Importantly, as a follow up study to a previous report,⁴⁸ in an evaluation of asbestos-exposed workers who developed colon cancer, 38% were found to have asbestos fibers and/or bodies present in their colon tissue, while in contrast 20 unexposed colon cancer patients had no asbestos fibers and/or bodies found.⁴⁹

Human Epidemiologic Studies:

There have been a number of epidemiologic studies over the past decades. The presence of asbestos-induced pleural plaques increased the risk of colorectal cancer among men occupationally exposed to asbestos, especially those with evidence of nonmalignant asbestos-associated radiographic changes.⁵⁰ A Canadian study demonstrated an association with a number of occupations and industries, including exposure to asbestos and other pollutants, with excess colorectal cancer risk.⁵¹ A study of exposed subjects in Normandy, France, concluded that their study “confirmed the established relationship between asbestos exposure and pleuro-pulmonary and peritoneal cancers, our study also suggests a causal relationship between asbestos exposure and colorectal cancer.”⁵² While these and a number of other studies have reported association of asbestos exposure and colorectal cancer risk, some studies, such as one conducted in southeast Michigan, have not found such an association,⁵³ nor a meta-analysis conducted by Weiss in 1995.⁵⁴ The discrepant results of the studies could be attributed to varying factors between the studies, including amounts of the asbestos exposure and methodological differences.

Importantly, the Weiss meta-analysis was conducted 25 years ago and did not include many pertinent reports in the interim. Three new meta-analyses have since been reported. The first, by Oddone et al, evaluated manuscripts published from 1960 to 2013, and included only prospective, case-control and meta-analysis studies were as eligible for this study, and the articles had to report at least one risk or mortality estimate to be included in quantitative analysis, [standardized mortality ratio; standardized incidence ratio; hazard ratio; RR; odds ratio (OR)] and a precision estimate (95%CI) relating exposure to an industrial branch to colon, rectal, or colorectal cancer or enough data to calculate them.⁵⁵ They found that workers in the sector of repair and installation of machinery exposed to asbestos were at increased risk of colorectal cancer (RR = 1.40, 95%CI: 1.07-1.84).⁵⁵

A second even more recent meta-analysis by Kwak et al,⁵⁶ evaluated 46 studies up to 4/2/2018, which was updated to exclude 7 redundant cohorts leaving 39 total studies.^{57,58} The inclusion criteria were as follows: studies of workers exposed to asbestos, cohort studies and studies that reported mortality data for colorectal cancer (SMR or provision of data enabling calculation of the SMR). They excluded studies based on the following criteria: those that did not clearly define asbestos exposure; studies of environmental exposure to asbestos; those that reported only incidence data for colorectal cancer; studies that provided quantitative risk estimates other than the SMR (eg, RR, proportional mortality ratio, OR, HR); meta-analyses and reviews; and studies that could not be found. They concluded that occupational exposure to asbestos was significantly associated with colorectal cancer with an overall pooled standardized mortality ratio of 1.16 (95% CI: 1.05 to 1.29), that remained statistically significant even after excluding the seven overlapping studies in the reanalysis, with an overall pooled standardized mortality ratio of 1.16 (95% CI: 1.03 to 1.29).^{57,58} They also concluded: “The pooled SMR for colorectal cancer was elevated in studies in which the asbestos-associated risk of lung cancer was also elevated (1.43; 95% CI: 1.30 to 1.56). This implies that the risk of colorectal cancer mortality increases as the level of asbestos exposure rises. A sensitivity analysis showed robust results and there was no publication bias. Although the effect size was small and the heterogeneity among studies was

large, our findings indicate that occupational exposure to asbestos is a risk factor for colorectal cancer.”

Yet another third meta-analysis by Huang et al,⁵⁹ including 47 cohorts prior to July 2017 was recently reported. The following was their methodology, “inclusion criteria for the literature that was selected for analysis included asbestos as a clear exposure factor; standardized mortality ratio, standardized incidence ratio and hazard ratio record included, research method is a cohort study. Exclusion criteria of the literature: repeated articles or data, animal experiment data; review of records that were not original; incomplete data information; only the newest or most informative article of the same cohort.” They observed and concluded, “The overall colorectal cancer SMR for synthesis cohort was 1.07 (95% CI 1.02–1.12). Statistically significant excesses were observed in exposure to mixed asbestos (SMR/SIR=1.07), exposure to production (SMR/SIR=1.11), among asbestos cement workers (SMR/SIR=1.18) and asbestos textile workers (SMR/SIR=1.11). Additionally, we determined that the SMR for lung cancer increased with increased exposure to asbestos, as did the risk for colorectal cancer. This study confirms that colorectal cancer has a positive weak association with asbestos exposure.”

An important epidemiologic study not included in any of the above meta-analyses was conducted in French men.⁶⁰ The abstract from this study is as follows: “Volunteer retired workers previously exposed to asbestos were invited to participate in the “French Asbestos-Related Diseases Cohort (ARDCo)” screening program between 2003 and 2005. Additional data on risk factors for colorectal cancer were collected from the ARDCo subsample of 3,769 participants in 2011. Cases of colon and rectal cancer were ascertained each year through 2014 based on eligibility for free medical care following a cancer diagnosis. Survival regression based on the Cox model was used to estimate the relative risk of colon and rectal cancer separately, in relation to the time since first exposure (TSFE) and cumulative exposure index (CEI) to asbestos, and with adjustment for smoking in the overall cohort and for smoking, and certain risk factors for these cancers in the ARDCo subsample. Mean follow-up was 10.2 years among 14,515 men, including 181 colon cancer and 62 rectal cancer cases (41 and 17, respectively, in the ARDCo-Nut subsample). In the overall cohort, after adjusting for smoking, colon cancer was significantly associated with cumulative exposure (HR = 1.14; 95% CI: 1.04 to 1.26 for a 1-unit increase in ln-CEI) and ≥ 20 -40 years since first exposure (HR = 4.67; 95% CI: 1.92, 11.46 vs. 0-20 years TSFE) and inversely associated with 60 years TSFE (HR = 0.26; 95% CI: 0.10, 0.70). Although rectal cancer was also associated with TSFE 20-40 years (HR = 4.57; 95% CI: 1.14, 18.27), it was not associated with ln-CEI, but these findings must be interpreted cautiously due to the small number of cases.” The authors of this report concluded, “Our findings provide support for an association between occupational exposure to asbestos and colon cancer incidence in men.”

In another epidemiologic study not included in the above meta-analyses, the Netherlands Cohort Study, a large cohort ($n = 58,279$ men, aged 55–69 years at baseline) was studied specifically addressing risk differences between relatively low and high exposure to asbestos, risk associated with cancer subtypes, the influence of potential confounders and the interaction between asbestos and smoking in relation to cancer risk.⁶¹ Asbestos exposure was estimated by linkage to a job-exposure matrix. After 17.3 years of follow-up, 187 esophageal, 486 gastric and 1,724 colorectal cancer cases were available for analysis. This prospective population-based study showed that asbestos exposure was associated with overall gastric cancer, EAC, GNCA, total and distal colon cancer and rectal cancer.

Weighing the body of evidence available in totality, as delineated above, it is my opinion that asbestos causes colorectal cancer.

8. **Cigarette smoking and Colon Cancer:** Cigarette smoking is a known carcinogen for many cancer types. This includes colorectal cancer. Recent studies demonstrated association of the amount and duration of smoking with colorectal cancer in Korea and Norway.^{26,62} Asbestos exposure acts synergistically with cigarette smoking to increase the risk of developing lung cancer 60 times over that of a similarly matched non-smoking, non-asbestos-exposed cohort. A total of 925 colorectal cancer cases and 2775 controls were included in the analysis. Odds ratios (OR) and 95% confidence intervals (CI) were computed by logistic regression models adjusting for potential confounders. In men, the risk of colorectal cancer significantly increased for heavy smokers who smoked ≥ 40 pack-years (OR 1.74, 95% CI 1.22-2.50), ≥ 40 years (OR 1.50, 95% CI 1.05-2.16), or ≥ 40 cigarettes/day (OR 1.92, 95% CI 1.04-3.54). Men showed a significant increase in risk, especially for rectal cancer with an increasing amount or duration of smoking. In women, distal colon cancer risk increased in smokers who smoked ≥ 20 years (OR 3.21, 95% CI 1.27-8.14) or ≥ 20 cigarettes/day (OR 4.75, 95% CI 1.09-20.57). Additionally, female smokers who smoked ≥ 20 cigarettes/day had an increased risk of rectal cancer (OR 6.46, 95% CI 1.64-25.46). Regarding the association of cigarettes smoked per day and the risk of rectal cancer, there was no significant difference between men and women (gender interaction p value = 0.14). Mechanistic studies demonstrating causation have demonstrated that in colon cancer cell lines, treatment with nicotine increased COX-2 expression and the release of its enzymatic product PGE₂. Moreover, nicotine-stimulated cells showed increased migratory and invasive behavior, mesenchymal markers up-regulation and epithelial markers down-regulation, nuclear translocation of the β -catenin, increase of MMP-2 and MMP-9 activity, and enhanced NF- κ B expression.⁶³ Also, smoking is known to influence messenger RNA expression in colorectal cancers. As recent study examined current smoking, current versus never and former versus never smoking, and pack-years smoked with miRNA expression in normal mucosa as well as differential miRNA expression between paired normal and carcinoma tissue for colon and rectal tissue to determine associations between smoking and miRNA expression.⁶⁴ The study results suggested that cigarette smoking can alter miRNA expression and, given associations with CIMP high and MSI tumor molecular phenotype, it is possible that smoking influences tumor phenotype through altered miRNA expression.

Second hand smoke is also a well-recognized risk factor for cancers, including cancers other than the lung.⁶⁵ Studies have demonstrated an association of passive smoke exposure and colorectal cancer.^{66,67}

Smoke and asbestos exposure together accentuates the risks of developing cancers.⁶⁸⁻⁷¹

9. **Details of Mr. Rodolfo Rodriguez' colorectal cancer and prognosis:** At age 66, Mr. Rodriguez was diagnosed with a recto-sigmoid mass on 9/15/16, and biopsy confirmed *KRAS* mutated invasive adenocarcinoma. *KRAS* is an oncogene that is often mutated, and consequently overactive, in colorectal cancer. CT scan on 9/15/16 revealed metastatic disease to retroperitoneal para-aortic lymph nodes and also right pelvic sidewall invasion, along with seminal vesicle and prostate invasion. Therefore, he was staged as stage IV at first diagnosis. He received palliative therapy but ultimately, he died due to this cancer on 10/10/17. I am still awaiting complete medical records to opine on his exact treatment history.

Factors that, more likely than not, each contributed to the development of his colorectal cancer include his exposure to asbestos, his documented obesity, his smoking and his diabetes. He did not have a known inherited family risk of cancer. He did not have a sedentary lifestyle as far as I can tell from the records.

I may also provide an opinion on the reasonableness or the cost of Mr. Rodriguez's cancer treatment and its necessity. I am waiting for said billing records and will amend my report if necessary.

10. **Conclusions:** Taking everything above into account, Mr. Rodolfo Rodriguez was exposed to asbestos during his time working at Southern Pacific Railroad Company. Asbestos is a known risk factor for cancer, and more likely than not was contributory to his development of his colorectal cancer, which was stage IV at the time of his diagnosis. As such, it also contributed to his death. He was treated appropriately with palliative intent for his cancer, yet as would be expected for stage IV colorectal cancer, he ultimately succumbed to his disease.

My opinions are based on medical fact and given with reasonable medical certainty. I rely on my experience in treating this and other gastrointestinal cancers, along with leading journals and reports in the field.

Best Regards,

A handwritten signature in black ink, appearing to read 'D Catenacci', followed by a vertical line.

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Documents Considered/Provided

Please also see expert report for additional documents that are cited.

MEDICAL RECORDS

Alpine Medical Center
Big Bend regional Medical Center
Midland Memorial Hospital
Marfa County Clinic
Death Certificate
DX Laboratory Path Report
Courtney Crim, MD Report

DEPOSITIONS

Rito Ortega
Daniel Rodriguez
Diana Rodriguez
Jose Rodriguez
Rosa Maria Rodriguez

OTHER

Richard Miller Report
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SCIENTIFIC ARTICLES

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PERSONAL

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Place of Birth: Sarnia, Ontario, Canada
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EDUCATION:

1995-1999 Honors Bachelor of Science, BSc. University of Waterloo, Waterloo, Ontario, Canada.
1999-2003 Doctor of Medicine, MD. Wayne State University, Detroit, Michigan.
2011-2014 Master of Science in Health Studies, MSc University of Chicago, Chicago, Illinois.
Biostatistics, Clinical & Translational Investigation.

POSTDOCTORAL TRAINING:

2003-2006 Internal Medicine Intern/Resident
UCLA Medical Center, Los Angeles, California

2006-2007 Clinical Fellow, Medical Oncology/Hematology
University of Chicago Medical Center, Chicago, Illinois.

2007-2010 GI Translational Research Fellow, Digestive Malignancies Laboratory
PI: Ravi Salgia. University of Chicago Medical Center, Chicago, Illinois.

POSTDOCTORAL EDUCATIONAL WORKSHOPS:

2007 AACR "Molecular Biology in Clinical Oncology" Workshop
Given Institute of the University of Colorado, Aspen, Colorado. July 1-7

2007-2008 Clinical Research Training Program, Essentials of Patient Oriented Research (EPOR I)
University of Chicago Medical Center, Chicago, Illinois. **Fall & Winter**

2008 Summer Workshops in Molecular Biology, New England Biolabs
Smith College, Clark Science Center, Northampton, MA. July 6-19.

2008 ECCO-AACR-ASCO "Methods in Clinical Cancer Research"
Flims, Switzerland, June 21-27.

2017 AAI "Advanced Course in Immunology" Boston, MA. July 23-28.

ACADEMIC APPOINTMENTS

2010-2012 Instructor, Department of Medicine, Section of Hematology/Oncology, University of Chicago, IL

2010- Member, Comprehensive Cancer Research Center, University of Chicago, IL

2012-2018 Assistant Professor, Department of Medicine, Hematology/Oncology, University of Chicago, IL

2016-2018 Associate Director, Gastrointestinal Oncology Program

2018- Director, Interdisciplinary Gastrointestinal Oncology Program

2018- Assistant Director, Translational Research, Comprehensive Cancer Center

2019- Associate Professor, Department of Medicine, Hematology/Oncology, University of Chicago, IL

HOSPITAL APPOINTMENTS

2010- Attending Physician. University of Chicago Medical Center, Chicago, IL.

LICENSURE AND CERTIFICATION:

Licensed to practice medicine:

05/2005-2009 California: #A91242
07/2006- Illinois: #036-115556
American Board of Internal Medicine:

Daniel Catenacci, M.D.

2006-2016 Internal Medicine
2009-2019 Medical Oncology (Hematology: board eligible)

PROFESSIONAL MEMBERSHIPS and ACTIVITIES:

1999-2003 American Medical Association
2003-2004 American College of Physicians
2002- The Pharos, Alpha Omega Alpha AΩA quarterly
2005- Medical Council of Canada
2006- American Society of Clinical Oncology, Associate Member
2006- American Society of Hematology, Associate Member
2007- American Association for Cancer Research, Associate Member
2010- Associate Investigator Pharmacogenomics and Experimental Therapeutics
2010- University of Chicago Comprehensive Cancer Center Member
2013- American Gastroenterological Association, Associate Member
2015- Overseas Fellow of the Royal Society of Medicine, United Kingdom

HONORS AND AWARDS:

1995-1999 **University of Waterloo**, Waterloo, Ontario, Canada, (Undergraduate):

- Dean's Honors List, Undergraduate Year I to Year IV.
- Nominated for the Governor General's Silver Medal and Alumni Gold Medal for highest academic standing in Faculty of Science, 1999
- Recipient of Sony of Canada Science Scholarship for highest academic standing, Faculty of Science, University of Waterloo, 1998

1999-2003 **Wayne State University** School of Medicine, Michigan (Medical School):

- Honors with Highest Distinction (*Summa Cum Laude*), Years I to IV.
- *Alpha Omega Alpha* AΩA Honor Medical Society, Inducted Yr II, 2001

1999-2003 **Harvard Medical School**/Harvard Institute of Medicine (Medical School):

- William F. von Liebig Summer Research Fellowship, Summer, 2000

2003-2006 **UCLA Medical Center** (Residency):

- Distinguished Teacher Award for UCLA Interns and Medical Students, 2004-2006

2006-2010 **University of Chicago** Medical Center (Fellowship):

- ASCO 2009 Young Investigator Award (YIA), 07/2009-06/2010.
- Amgen Hematology & Oncology Fellowship Grant Support Program, 04/2008-03/2009

2010-2012 **University of Chicago** Medical Center (Instructor):

- Cancer Research Foundation Young Investigator Award (CRF YIA). 10/2010-09/2011.
- K-12 Scholar. Paul Calabresi Career Development in Clinical Oncology. 10/2010-09/2013.
- NCI/CTEP Career Development LOI Awarded – A Randomized Discontinuation Trial of OSI-906 in metastatic Colorectal Cancer After Two or More Lines of Prior Therapy 10/29/2010.

2012-2018 **University of Chicago** Medical Center (Assistant Professor):

- ALLIANCE for Clinical Trials in Oncology Foundation Young Investigator Award 07/2012-06/2013
- Esophago-Gastric NCI Task Force, ALLIANCE Junior Member (06/2012-present)
- Best Abstract and Oral Presentation at the 5th Annual WIN (Worldwide Innovative Networking in Personalized Cancer Medicine) Symposium. July 7-10, 2013. Paris, France.
 - Towards personalized treatment for gastroesophageal adenocarcinoma: strategies to address inter- and intra- patient tumor heterogeneity: PANGAEA
- Best Abstract Translational Research Faculty Category Annual Janet Rowley Research Day University of Chicago, March 4, 2014 PANGAEA Clinical trial Design and Pilot results.
- K23 Scholar Awarded 9/2014-8/2017.
- Named on the "Chicago's Top Cancer Doctors' List. December, 2016
- Tree of Life Medical Award. Debbie's Dream Foundation for Stomach Cancer. April, 2018

2019- **University of Chicago** Medical Center (Associate Professor):

CLINICAL

I am an adult Medical Oncologist with sub-specialization in Gastrointestinal Cancers, with focus on upper GI cancers, and special interest in Esophagogastric adenocarcinoma and Cholangiocarcinoma/Gallbladder cancer.

Daniel Catenacci, M.D.

2010- GI Oncology Clinic (1 day/week, 12 months/year, **30% effort**)

2010- Inpatient Service - Chemotherapy service, Housestaff Supportive Oncology (4 weeks/year, **10% effort**)

SCHOLARSHIP:

BIBLIOGRAPHY:

Peer Reviewed Articles:

Original Articles

1. Zhang W, **Catenacci DVT**, Duan S, Ratain MJ. A Survey of the Population Genetic Variation in the Human Kinome. *J of Hum Genet.* Aug;54(8):488-92. 2009. PMID 19644514.
2. **Catenacci DVT**, Cervantes G, Yala S, Nelson EA, El-Hassani E, Kanteti R, El Dinali M, Hasina R, Brägelmann J, Seiwert T, Sanicola M, Henderson L, Grushko T, Olopade O, Karrison T, Bang YJ, Kim WH, Tretiakova M, Vokes EE, Frank DA, Kindler HL, Huet H, Salgia R. RON (*MST1R*) is a novel prognostic marker and therapeutic target for gastroesophageal adenocarcinoma. *Cancer Biol Ther.* 12:1, 1-38, July 1, 2011. PMID 21543897. **Featured Article**.
3. **Catenacci DVT**, Henderson L, Xiao SY, Priti Hegde, Premal Patel, Robert L. Yauch, Peterson A, Salgia R. Durable complete response of gastric cancer with anti-MET therapy followed by resistance at recurrence. *Cancer Discov.* 2011 Dec 1;1(7):573-579.PMID: 22389872.
4. Kanteti R, Krishnaswamy S, **Catenacci DVT**, Cervantes G, Henderson L, Tan Y, El-Hassani E, Husain AN, Tretiakova M, Huet R, Salgia R. Differential Expression of RON in Small and Non-Small Cell Lung Cancers. Epub May24 *Genes Chromosomes & Cancer* 2012. PMID 22585712.
5. Shah MA, Wainberg ZA, **Catenacci DVT**, Hochster HS, Ford J, Kunz P, Lee FC, Kallender H, Cecchi F, Rabe DC, Keer H, Martin AM, Liu Y, Gagnon R, Bonate P, Liu L, Gilmer T, Bottaro DP. Phase II Study Evaluating 2 Dosing Schedules of Oral Foretinib (GSK1363089) in patients with Advanced or Metastatic Gastric Cancer. *PLoS One.* 2013;8(3):e54014. PMID:23516391
6. Geynisman DM, Zha Y, Kunnavakkam R, Aklilu D, **Catenacci DVT**, Polite BN, Rosenbaum A, Namakydoust A, Karrison T, Gajewski TF, Kindler HL. A randomized pilot phase I study of modified Carcinoembryonic antigen (CEA) peptide (CAP1-6D)/Montanide/GM-CSF-vaccine (CEA-vac) in patients (pts) with pancreatic adenocarcinoma (PC). *Journal for ImmunoTherapy of Cancer* 2013, **1**:8. PMID pending
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8. Salgia R, Patel P, Bothos J, Yu W, Bai S, **Catenacci DVT**, Peterson A, Ratain M, Polite B, Mehnert J, Moss R. Phase I dose-escalation study of onartuzumab as a single agent and in combination with bevacizumab in patients with advanced solid malignancies. *Clin Cancer Res.* 2014 Feb 27. PMID:24493831.
9. **Catenacci DVT**, Liao WL, Thyparambil S, Henderson L, Peng Xu, Rambo B, L Zhao, Hart J, Xiao SY, Bengali K, Uzzell J, Dafler M, Krizman D, Cecchi F, Bottaro D, T Karrison, Veenstra TD, Hembrough T, Burrows J. Absolute Quantitation of c-Met using Mass Spectrometry for Clinical Application: Assay Precision, Stability, and Correlation with *MET* gene amplification in FFPE Tumor Tissue. *PLoS One.* 2014 Jul 1;9(7):e100586. PMID:24983965.
10. **Catenacci DVT**, Amico A, Nielsen S, Geynisman D, Carey GB, Gulden C, Fackenthal J, Kindler HK, Olopade F. Tumor Genome Includes Germline Genome - Are We Ready For Surprises? *International Journal of Cancer* 2014 August 2014. PMID 25123297.
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12. Ali SM, Sanford EM, Klempner SJ, Robinson DA, Wang K, Palma NA, Chmielecki J, Yelensky R, Palmer GA, Morosini D, Lipson D, **Catenacci DVT**, Braithe F, Erlich R, Stephens PJ, Ross JS, Ou SH, Miller VA. Prospective Comprehensive Genomic Profiling of Advanced Gastric Carcinoma Cases Reveals Frequent Clinically Relevant Genomic Alterations and New Routes for Targeted Therapies. *Oncologist.* 2015 Apr 16. PMID 25882375

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15. **Catenacci DVT**, Bahary N, Edelman M, Nattam S, Brockstein B, Sparano J, Kozloff M, Cohen D, Stiff P, Sleckman B, Thomas S, Lenz H, Henderson L, Vannier M, Karrison T, Stadler WM, Kindler HL. Final Analysis of a Phase Ib/Randomized Phase 2 Trial of Gemcitabine plus Placebo or Vismododib (GDC-0449), a Hedgehog Pathway Inhibitor, in Patients with Metastatic Pancreatic Cancer. *J Clin Oncol* 2015 2015 Nov 2. [Epub ahead of print]. PMID: 26527777
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36. Hong DS, LoRusso P, Hamid O, Janku F, Kittaneh M, **Catenacci DVT**, Chan E, Bekaii-Saab T, Gadgeel SM, Loberg RD, Amore B, Hwang Y, Tang R, Ngarmchamnanrith G, Kwak EL. Phase I Study of AMG337, a Highly Selective Small Molecule MET Inhibitor, in Patients With Advanced Solid Tumors. *CCR* 2018. PMID 30425090

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38. Bang YJ, Muro K, **Catenacci DVT**, Garrido M, Koshiji M, Dalal R, Wainburg Z, Fuchs C, Kang Y. Pembrolizumab For the First-Line Treatment of Patients With Advanced Gastric or Gastroesophageal Junction Adenocarcinoma: Results From Cohorts 2 and 3 of the KEYNOTE-059 Study. *In press Gastric Cancer* 2018.

Editorials/Commentaries:

39. Khoury J, **Catenacci DVT**. Next-generation companion diagnostics: Promises, Challenges, and Solutions. *Archives of Pathology & Laboratory Medicine* Aug 28 2014. PMID 25166874.
40. **Catenacci DVT**. The Expansion Platform Type II Design: Testing a Treatment Strategy. *Epub Sept 8 2015 Lancet Oncology*. 26342235
41. Zhang SQ, **Catenacci DVT**. How can next-generation diagnostics aid pancreatic adenocarcinoma treatment? *Future Oncology* Mar 2016. 26831761
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44. Klempner SJ, **Catenacci DVT**. Variety is the Spice of Life, But Maybe Not in Gastroesophageal Adenocarcinomas. *Cancer Discovery* 2019 PMID 30737213.
45. Rajagopal P, **Catenacci DV**, Olopade OO. The time for mainstreaming germline testing for breast cancer patients is now. *J Clin Oncol* 2019 *In press*

Reviews:

46. **Catenacci DVT**, Schiller GJ. Myelodysplastic Syndromes: A Comprehensive Review. *Blood Reviews* Nov 2005; 19(6):301-319. PMID: 15885860 **Top 20 cited in *Blood Reviews* towards the 2007 impact factor.
47. **Catenacci DVT**, Kozloff M, Kindler HL, Polite B. Personalized Colon Cancer Care in 2010. *Semin Oncol*. 2011 Apr;38(2):284-308. PMID:21421118
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49. Geynisman DM, **Catenacci DVT**. Toward Personalized Treatment of Advanced Biliary Tract Cancers. *Discov Med*. 2012 Jul;14(74):41-57. PMID: 22846202.
50. Sehdev A, **Catenacci DVT**. Perioperative Therapy for Locally Advanced Gastroesophageal Cancer: Current Controversies and Consensus of Care. *Journal of Hematology & Oncology*. Sept 2013, 6:66. PMID: 24010946
51. Sehdev A, **Catenacci DVT**. Gastroesophageal Cancer: Focus on Epidemiology, Classification and Staging. *Discov Med*, September 2013 16(87):103-111. PMID: 23998446.
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53. Maron SB, **Catenacci DVT**. Update on Gastroesophageal Adenocarcinoma Targeted Therapies. *Epub*

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Hematol Oncol Clin North Am 2017. PMID: 28501091

54. Joshi SS, Maron SB, **Catenacci DVT**. Pembrolizumab for treatment of advanced gastric and gastroesophageal junction adenocarcinoma. Nov 2. doi: 10.2217/fon-2017-0436. [Epub ahead of print] 2017 *Future Oncology*. PMID: 29094609

Book Chapters:

55. **Catenacci DVT**, Cohen E., Villaflor V. Gastroesophageal Tumors: Principles and Practice. Chapter 33: Principles of Multimodality Therapy, pages 229-242. Mar 2009. (Edited by Jobe, Hunter and Thomas).
56. **Catenacci DVT**. Cancer Biology Review: A Case-Based Approach. Chapter 4: Cell Surface Receptors and Signal Transduction: Principles of Cancer Biology. 2014 (Edited by Stadler and Winters).
57. Polite BN, **Catenacci DVT**. ASCO Self Evaluation Program (SEP) 7th edition, Gastrointestinal Malignancies Chapter. 2021

Original Articles under revision, submitted or in preparation:

58. Falchook G, Kurzrock R, Amin H, Fu Siqing, Piha-Paul S, Janku F, Eskandari G, **Catenacci DVT**, Klevesath M, Bruns R, Stammberger U, John A, Hong D. First-in-Human Phase I Trial of the Selective c-Met Inhibitor Tepotinib in Patients with Advanced Solid Tumors. *Manuscript submitted*.
59. Oliwa T, Maron SB, Chase L, Lomnicki S, Catenacci DVT, Furner B. Obtaining knowledge in pathology reports through a natural language processing approach with classification, named-entity recognition and relation-extraction heuristics. *Manuscript submitted*.
60. Maron SB, Lomnicki S, Chase L, Joshi S, Nagy B, Lanman R, Lee J, **Catenacci DVT**. 'Circulating tumor DNA sequencing of gastroesophageal adenocarcinoma.' *Manuscript submitted*.
61. Parikh AR, He Y, Hong T, Corcoran R, Clark J, Ryan D, Zou L, Ting D, **Catenacci DVT**, Chao J, Fakih M, Klempner SJ, Ross JS, Frampton GM, Miller VA, Ali SM, Schrock AB. Analysis of DNA damage response gene alterations and tumor mutational burden across 17,486 tubular GI carcinomas: Implications for therapy. *Manuscript in preparation*.
62. **Catenacci DVT**, Chao J, Klempner S, Janjigian Y, Kim R, Liepa A, Kuder C, Chin S, Shah M, Fuchs C. A Systematic Review of First and Second Line Randomized Controlled Trials for Advanced Gastroesophageal Adenocarcinoma: Towards a Treatment Sequencing Strategy. *Manuscript in Preparation*
63. **Catenacci DVT**, Karrison T, Dignam J, Ji J. Statistical considerations of the 'Expansion Platform Clinical Trial Design Type II'. *Manuscript in preparation*.
64. Kanteti R, Maron SB, Tumuluru S, Chase L, Henderson L, **Catenacci DVT**. Targeted therapies for targeted populations: Met inhibition for *MET* amplified gastroesophageal adenocarcinoma. AMG DN Merck Serono TL *Manuscript in Preparation*

RESEARCH SUPPORT:

Current Grant Support:

R01 5R01CA132897-07: Bayesian Inference for Tumor Heterogeneity with Next Generation Sequencing Data from PANGEA. PI Yuan Ji (Biostatistician Northshore/University of Chicago. (2015-2020) (**Catenacci 10%**)

Endoscopic Research Award 2018 (PI Chapman) "Liquid Biopsies of the Portal Vein Using Endoscopic Ultrasound for Next Generation Sequencing of circulating tumor DNA for Therapeutic and Prognostic Stratification in Pancreatic Cancer." (2018-2019) (\$60,000, 0% effort).

General Research Fund: 2010-

Ullman Scholar Award: "Evaluation of intratumoral tumor and immune cell heterogeneity" (7/2018-6-/2019 \$50,000, 0% effort).

Live Like Katie Foundation Award: 2013- (\$300,000, 10% effort)

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Sal Ferrara Fund for PANGAEA Award: 2014- (\$300,000, **10% effort**)

Castle Foundation Award: 12/31/16-12/31/2020 (\$250,000 over 4 years, **15% effort**)

Submitted/Planned Submission, Pending Grant Support:

Submitted: R01: Modelling the Population Health Impact of Cancer Genomic Technologies by Race and Ethnicity. PI Fabrice Smieliauskas. (2018-2023, Catenacci **effort 5%**)

Submitted: R01: Integration of genomic and phenotypic data for cancer research and clinical support. PI Yitan Zhu, Northshore. (2018-2023, Catenacci **effort 10%**)

Submitted: R01 PAR-18-654: Race-specific Regulation of Gene Networks. PI Isadore Ragoutsos (Jefferson). (2019-2023, Catenacci **effort 10%**).

Submitted: Genentech Collaborative Funding: Towards Personalized Immune and Targeted Therapies for Gastroesophageal Adenocarcinoma. PI Catenacci (\$250,000 over 3 years, **25% effort**)

Submitted: Translational Team Science Award W81XWH-18-PRCRP-TTSA DOD. Characterizing genomic evolution and the development of resistance to kinase therapy in gastric adenocarcinoma. PIs Catenacci/Bass. (UoC 810K over 4 years; PI Catenacci **20% effort**).

Planned R01: Tumor Molecular and Immunologic Biomarker Heterogeneity in Gastroesophageal Adenocarcinoma. PI Catenacci **20% effort** Plan 2/5/2018 cycle

Planned: R01: Targeting Wild-Type Amplified KRAS in Gastroesophageal Adenocarcinoma. PI Catenacci **20% effort** Plan 2/5/2018 cycle

Past Grant Support:

Amgen Hematology & Oncology Fellowship Grant. "The Role of RON Receptor Tyrosine Kinase in Gastroesophageal Cancers" (7/2008-06/2009).

CTSA-ITM Core Subsidies Fellow Grant. "Immunohistochemical Evaluation of The Role of RON and MET Receptor Tyrosine Kinases in Gastroesophageal Cancers" (1/09-06/09).

R21. "Novel Targeted Therapy in Pancreatic Cancer". Co-PI Salgia/Kindler (07/2009-06/2011).

ASCO 2009 Young Investigator Award. "The Role of RON (MST1R) Receptor Tyrosine Kinase in Gastroesophageal Cancers as a Therapeutic Target." (07/09-06/10).

Cancer Research Foundation Young Investigator Award (CRF YIA). "The Role of RON Tyrosine Kinase in Gastroesophageal Cancer". \$75,000 5% effort (1/2011-12-2011).

American Research and Recovery Act (ARRA) NCI 8418. "GDC-0449 for Pancreas" A Randomized phase 2 trial of gemcitabine plus GDC-0449, a Hh pathway inhibitor, in metastatic pancreatic cancer.

PI Salgia/Kindler. (07/09-06/13). **Laboratory Correlates** PI Catenacci. \$35,000. 0% effort.

ALLIANCE/CALGB for Clinical Trials in Oncology Foundation YIA 07/2012-06/2013. "Laboratory Correlates Companion Study for CALGB 80101 Evaluating MET, RON, HER2, TOP2A and ERCC1 as Biomarkers for Gastroesophageal Adenocarcinoma." \$30,000, 0% effort (07/1/12 – 6/2014)

K-12. Paul Calabresi Clinical Oncology Career Development K12 Program. "The Role of RON Tyrosine Kinase in Gastroesophageal Cancer". \$125,500/yr (10/1/10 – 9/30/13, 75% effort)

"The role of RON tyrosine kinase in relation to targeted MET inhibition in gastroesophageal cancer." OSI Pharmaceuticals. 04/23/2012 – 04/22/2014 (\$140,000, 5% effort)

OncoPlex Diagnostics Collaborative Funding: OncoPlex Dx Project ID: Work Orders 3,4,6 (\$24,500 each, 2012-2014, 0% effort)

UCCCC Pilot Precision Medicine Award. "Towards Personalized Treatment of Gastroesophageal Adenocarcinoma: A Pilot Trial of PANGAEA" 01/01/14-12/31/14 (\$35,000, 0% effort)

Amgen Collaborative Funding: Evaluation of MET expression and gene copy number in gastroesophageal tissues. 09/09/2013 – 09/08/2015 (\$183,500, 0.5% effort)

ITM Pilot Award: Exhaustive detection of drug resistance mutations. 9/2015-9/2016 (\$35,000, 0% effort). PI Chung-I Wu/Catenacci DVT.

K23. PANGAEA-IMBBP Pilot Trial (Personalized Antibodies for GastroEsophageal Adenocarcinoma Pilot Trial). 9/11/14-8/31/17 (\$156,000/yr, 75% effort).

PI Daniel Catenacci.

Genentech Collaborative Funding: Towards Personalized Therapy of Gastroesophageal Adenocarcinoma. (\$250,000 10/2014-10/2017, 10% effort)

OncoPlex Diagnostics Collaborative Funding: OncoPlex Dx Project ID: Work Order 7. (\$140,000/yr) (\$280,000 1/14/15-1/13/18, 0% effort)

ORAL PRESENTATIONS**Invited Speaking****International Meetings/Conferences**

May 25, 2008. “RON receptor tyrosine kinase: A novel therapeutic target of gastroesophageal adenocarcinoma.” Chinese National Genome Center, Shanghai, China.

May 26, 2008. “RON receptor tyrosine kinase: A novel therapeutic target of gastroesophageal adenocarcinoma.” First Peoples’ Hospital, Shanghai, China.

July 12, 2013 “Towards personalized treatment for gastroesophageal adenocarcinoma: strategies to address inter- and intra- patient tumor heterogeneity: PANGAEA”. WIN (Worldwide Innovative Networking in Personalized Cancer Medicine) 2013 5th Annual Conference. Paris, France. **Plenary Session ORAL Presentation & Best Abstract Award.**

<http://ecancer.org/conference/328-win-symposium-2013/video/2151/strategies-to-address-inter--and-intra--patient-tumour-heterogeneity--pangea.php>

<http://www.winsymposium.org/abstracts/abstract-publication-2/>

<http://www.winsymposium.org/program/program-at-a-glance/presentations-july-12/>

January 15, 2015 “Tumor Board: Management of Challenging Cases of Upper Gastrointestinal Cancers (ARS)” Invited Panelist. ASCO GI 2015, San Francisco, CA.

June 1, 2015 “Meeting Highlights: Gastrointestinal Cancer.” ASCO Trainee & Early-Career Oncologist Lounge. ASCO 2015, Chicago IL.

January 21, 2016 GI ASCO Oral Abstract Session – Discussant. ASCO GI 2016, San Francisco, CA.

January 21, 2016 “General Session 3: Multimodal Approaches for Advanced GE Junction Cancers (East and West)–Challenging Cases” Session Chair. ASCO GI 2016, San Francisco, CA.

June 29, 2017 GI ESMO Oral Abstract Session - Session VI Gastric Cancer LBA-009: **Catenacci DVT**, Wainberg Z, Fuchs CS, Garrido M, Bang YJ, Muro K, Savage M, Wang J, Koshiji M, Dalal RP, Kang YK. KEYNOTE-059 cohort 3: safety and efficacy of pembrolizumab (pembro) monotherapy for first-line treatment of patients (pts) with PD-L1-positive advanced gastric/gastroesophageal (G/GEJ) cancer. ESMO World GI July 28-30, 2017. *Barcelona Spain*. Oral Presentation, presented by Catenacci. Ann Oncol (2017) 28 (suppl_3): mdx302.008.

October 18, 2017 World CDx Annual Summit – Session: Clinical Implementation and Validation- “Improving Patient Recruitment and Retention on Precision Medicine Clinical Trials”. World CDx 9th Annual Summit Boston, MA.

December 14, 2017 “Perioperative systemic chemotherapy and the practical use of triplet for borderline resectable mCRC patients”. Chang Gung Memorial Hospital, Linkou District, Taiwan.

December 16, 2017 “Annual update on the treatment for metastatic colorectal cancer and its impact on personalized therapeutic approach.” Annual Meeting of the Society of Colon and Rectal Surgeons. Taipei, Taiwan.

June 19, 2018 “Gastroesophageal tumor molecular heterogeneity, molecular evolution, and implications in the clinic” Samsung Medical Center. Seoul, Korea.

June 21, 2018 Korean Cancer Association GI Gastric Cancer Plenary Session: “Overcoming tumor heterogeneity in Gastroesophageal Adenocarcinoma: the PANGAEA trial” Korean Cancer Association

Daniel Catenacci, M.D.

Annual Meeting. Seoul, Korea.

September 27, 2018 2nd Annual AACR Conference Translational Medicine, Session: Precision Cancer Medicine: "Implementing precision strategies to address molecular heterogeneity for gastroesophageal adenocarcinoma". Sao Paulo, Brazil.

May 9, 2019 13th International Gastric Cancer Congress, Session Novel Drugs (Non-immune therapy): "Personalized treatment: How to overcome tumor heterogeneity". Prague, Czech Republic.

September 9, 2019 "Overcoming tumor heterogeneity in Gastroesophageal Adenocarcinoma: the PANGEA trial." Eleventh International Workshop on Pharmacodynamics of Anticancer Agents. Monestier, France.

National

January 13, 2010 Visiting Professor Lecture: "RON is a novel prognostic marker and therapeutic target for gastroesophageal adenocarcinoma." Northwestern University, Chicago, IL.

March 12-14, 2010 "Novel therapies for gastroesophageal adenocarcinoma: A personalized treatment approach." World Congress on Gastroenterology & Urology. Marriott Omaha, USA.

June 21, 2011. "RON tyrosine kinase in cancer: no longer MET's Little Brother!" OSI/Astellas Pharmaceuticals. Farmingdale, Long Island, NY.

June 24, 2011. "RON tyrosine kinase in cancer: no longer MET's Little Brother!" AVEO Pharmaceuticals, Inc. Cambridge, MA.

October 5, 2011. "Targeted Therapies A New Generation of Cancer Treatments." OptumHealth's 20th Annual National Conference. Hyatt Regency in Minneapolis, MN.

October 20, 2011 "A Patient with Gastric Cancer Treated with a MET Inhibitor." Expert Forum on MET Inhibition. Oncology Network for Excellence. NCIR/CTEP, Bethesda, MD..

October 21, 2011. "Predicting Response to MET Targeted Agents with Biomarkers: *MET* Amplification." Expert Forum on MET Inhibition. Oncology Network for Excellence. NCI/CTEP, Bethesda, MD.

February 22-25, 2012. "MET tyrosine kinase: prognostic and predictive biomarkers of the MET pathway." 12th Annual Targeted Therapies of Lung Cancer Meeting. The Fairmont Miramar Hotel, Santa Monica, CA. Sponsored by the IASLC.

September 20, 2012. "Towards personalized treatment for gastroesophageal adenocarcinoma: strategies to address inter- and intra- patient tumor heterogeneity," Genentech, San Francisco.

October 25-26, 2012. "Gastrointestinal Cancer Overview: Gastroesophageal Adenocarcinoma, Colorectal Adenocarcinoma, Hepatocellular Carcinoma. FOCUS on MET Tyrosine Kinase." 2nd Annual Expert Forum on MET Inhibition. Oncology Network for Excellence. CTEP/NCI. Washington, DC.

October 25-26, 2012. "Gastroesophageal Adenocarcinoma: Strategies to address inter- & intra-patient tumor heterogeneity...a focus on MET." 2nd Annual Expert Forum on MET Inhibition. Oncology Network for Excellence. CTEP/NCI. Washington, DC.

October 25-26, 2012. "Colorectal Cancer: FOCUS on MET Tyrosine Kinase." 2nd Annual Expert Forum on MET Inhibition. Oncology Network for Excellence. CTEP/NCI. Washington, DC.

December 6, 2012. "Towards personalized treatment for gastroesophageal adenocarcinoma: strategies to address inter- and intra- patient tumor heterogeneity...PANGEA." AVEO Pharmaceuticals, Inc. Cambridge, MA.

Daniel Catenacci, M.D.

April 5, 2013. “Moderated Roundtable Discussion: Defining the major knowledge gaps and priorities for future research of cholangiocarcinoma”. Invited Panelist. CanLiv 3rd Annual Symposium: Harnessing Genomic-Driven Therapies for Hepatobiliary Cancers. Washington, DC.

May 18, 2013. “Treatment of Advanced Gastroesophageal Cancer: A Focus on Targeted Therapies” JACOB phase III Clinical Trial Investigators’ Meeting: A double-blind, placebo-controlled, randomized, multicenter Phase III Study evaluating the efficacy and safety of pertuzumab in combination with trastuzumab and chemotherapy in patients with HER2-positive metastatic gastroesophageal junction and gastric cancer. International Investigator Meeting. InterContinental, Chicago, IL.

June 20-22, 2013. “Gastroesophageal Adenocarcinoma in The Era of Targeted Therapies: A Focus on MET” Amgen Oncology Global Advisory Board. Thousand Oaks, CA.

May 4-6, 2014. Detection of Portal Vein (PV) Circulating Tumor Cells (CTCs) in Pancreatic Cancer (PC) patients obtained by EUS guided PV Sampling. A safety and Feasibility trial. Accepted as an **Oral Presentation**. Digestive Disease Week 2014. Chicago, IL.

November 24, 2014 Visiting Professor Lecture: “Strategies to address inter- and intra- patient tumor molecular heterogeneity using next-generation companion diagnostics and PANGEA: a novel clinical trial design”. Nantworks/NantHealth, Los Angeles, CA.

March 3, 2015. Visiting Professor Lecture: “Strategies to address inter- and intra- patient tumor molecular heterogeneity using next-generation companion diagnostics and PANGEA: a novel clinical trial design”. Grand Rounds University of California San Diego UCSD, CA.

March 5, 2015. Visiting Professor Lecture: “Strategies to address inter- and intra- patient tumor molecular heterogeneity using next-generation companion diagnostics and PANGEA: a novel clinical trial design”. Section of Oncology Weekly Meeting, Stanford University. Palo Alto, CA.

March 30/31, 2015. “Next-Generation Clinical Trials Incorporating Next-Generation Companion Diagnostics”. OMICS 2nd Annual Meeting, Patrick Soon-Shiong NantOmics. Los Angeles, CA.

July 16, 2015. “Next-Generation Clinical Trials Incorporating Next-Generation Companion Diagnostics”. FDA gastric cancer mini-symposium. Silver Spring, MD.

November 7, 2015 Debbie’s Dream Foundation for Stomach Cancer Inaugural Chicago Symposium. Committee Chair and Organizer, Moderator, Speaker (“Tumor Genomics, Immunotherapy, Clinical Trials, and Other Hopes for the Future”), O’Hare Marriot, Chicago IL.

November 14, 2015. “Tumors to the Liver: Metastatic Adenocarcinoma of Unknown Origin – Work up before Therapy and Role of Molecular Profiling to Sort it out” Hepatic Tumor Summit, Tampa FL.

November 14, 2015. “Does Molecular Profiling Predict Response to Therapy?” Hepatic Tumor Summit, Tampa FL.

June 17, 2016. Visiting Professor Lecture: “Tumor molecular heterogeneity, molecular evolution, and implications in the clinic.” Roswell Park Grand Rounds, Buffalo NY.

October 1, 2016. “Tumor Genome Analysis Includes Germline Genome!! Are We Ready For Surprises??” National Society of Genetic Counsellors 35th Annual Meeting. Seattle WA.

October 26, 2016. Visiting Professor Lecture: “Determining the Clinical Utility of Plasma ctDNA Next-Generation Sequencing”. Guardant Health. Redwood City, CA.

May 12, 2017. “Addressing Tumor Molecular Heterogeneity using A Novel Clinical Trial Design – PANGEA”. Symposium on Dose Selection for Cancer Treatment Drugs: Novel Clinical Trial Designs for Cancer Treatments. Stanford, Palo Alto, California.

Daniel Catenacci, M.D.

November 3, 2017. “KEYNOTE-059: Trial Efficacy and Safety of Pembrolizumab Alone or in Combination With Chemotherapy in Advanced Gastric or Gastroesophageal Cancer.” KEYNOTE-585 National Initiation Investigator Meeting, Hilton Dallas Lincoln Centre, Dallas, Tx.

January 16, 2018. Visiting Professor Lecture: “Intra-patient molecular heterogeneity a barrier to successful implementation of precision medicine in gastroesophageal adenocarcinoma – how to address?.” GI Oncology Grand Rounds University of California, San Francisco University of California San Francisco UCSF. San Francisco, CA.

February 13, 2018. Visiting Professor Lecture: “Tumor molecular heterogeneity, molecular evolution, and implications in the clinic.” Cancer Center Seminar, University of Texas Southwestern, Dallas, TX.

April 21, 2018. “Chemotherapy, Targeted Treatments, and Immunotherapy for Gastric and Esophageal Cancer: Hope for the Future”. Debbie’s Dream Foundation for Stomach Cancer 8th Annual Symposium and Live Webcast. Hollywood, FL.

August 4, 2018. “Best of ASCO 2018 : Gastrointestinal (Non Colorectal) Cancer”. Best of ASCO 2018, Denver, CO.

August 6, 2018. Visiting Professor Lecture: “Tumor molecular heterogeneity, molecular evolution, and implications in the clinic.” Cancer Center Seminar. Yale University Medical Center. New Haven, CT.

November 2, 2018. “Gastroesophageal Adenocarcinoma Overview of Epidemiology, Molecular Profiling and Treatment.” Phase III FIGHT study Investigator Meeting: A Study of Bemarituzumab (FPA144) Combined With Modified FOLFOX6 (mFOLFOX6) in Gastric/Gastroesophageal Junction Cancer (FIGHT). The Westin Austin Downtown. Austin, Tx.

January 12, 2019. “Tumor molecular heterogeneity, molecular evolution, and implications in the clinic – testing a treatment algorithm.” Cancer Center Showcase – Precision Oncology Applications and Utility at Cancer Centers Session. The Precision Medicine World Conference (PMWC) 2019. Santa Clara, CA.

Regional

March 13, 2009 “Targeting Hedgehog Signaling in Cancer,” The University of Chicago Phase II Consortium 14th Annual Symposium, Gleacher Center, Chicago, IL.

March 13, 2010. “Targeting Hedgehog Signaling in Cancer,” The University of Chicago Phase II Consortium 15th Annual Symposium, Gleacher Center, Chicago, IL.

May 6, 2011. “Perioperative Therapy for Gastroesophageal Adenocarcinoma”. The 3rd Annual Controversies in the Management of Complex GI Patients Symposium. The Ritz Carlton Hotel, Chicago, IL.

September 16, 2011. “A Laboratory Correlative Companion Study for CALGG 80101 evaluating MET, RON, HER2, TOP2A, and ERCC1 as Biomarkers for Gastroesophageal Adenocarcinoma” ACTION (Alliance for Clinical Trials In Oncology Group). Hyatt Regency O’Hare, Rosemont, IL.

Sept 15-18, 2011. “Developmental Therapeutics in Oncology: Updates from ASCO 2011 Best of ASCO Meeting”. The 14th Annual APAO Conference. APAO’s Best of ASCO Oncology Meeting. The Chicago Wyndham Hotel.

April 27, 2012. “Pancreatic Cancer: Hedgehog Signaling & the new era of FOLFIRINOX”. The University of Chicago Phase II Consortium 17th Annual Symposium, Gleacher Center, Chicago.

April 27, 2012. “Towards Personalized Cancer Care for Gastroesophageal Adenocarcinoma: Challenge, Controversy & Consensus,” The University of Chicago Phase II Consortium 17th Annual Symposium, Gleacher Center, Chicago.

September 07, 2012. “Systemic Therapy for Hepatocellular Carcinoma (HCC) and Biliary Tract Cancers,”

Daniel Catenacci, M.D.

The 4th Annual Gastrointestinal Cancer Symposium: Update on the Management of GI Cancer Patients. The Ritz Carlton Hotel, Chicago, IL.

April 12, 2013. "Towards personalized treatment for gastroesophageal adenocarcinoma: strategies to address inter- and intra- patient tumor heterogeneity...PANGAEA," The University of Chicago Phase II Consortium 18th Annual Symposium, Gleacher Center, Chicago.

October 10, 2014 "Personalized Colon Cancer Care: Are we there yet?" University of Chicago Symposium: "Colon, Rectum and Beyond: Innovations in Management of Inflammatory Bowel Disease, Colorectal Cancer and Pelvic Floor Disorders". The Board of Regents Room, American College of Surgeons, North St. Clair, Chicago, IL.

January 23, 2015 "Strategies to address inter- and intra- patient tumor molecular heterogeneity using next-generation companion diagnostics and PANGAEA: a novel clinical trial design". Grand Rounds Northshore Hospital, Chicago, IL.

April 13, 2015 "Strategies to address inter- and intra- patient tumor molecular heterogeneity using next-generation companion diagnostics and PANGAEA: a novel clinical trial design". NorthShore Scientific Society meeting, Chicago, IL.

November 5, 2015 "Analyzing Your Genome". Rolfe Foundation Symposium on Personalized Medicine. Cancer Wellness Center, Northbrook, IL.

April 8, 2016. "Tumor Molecular Heterogeneity, Molecular Evolution, and Implications in the clinic" The University of Chicago Phase II Consortium 21th Annual Symposium, Gleacher Center, Chicago.

November 7, 2017. "Personalized Medicine in the Gastrointestinal Oncology Clinic: Promises, Challenges, and Future Decisions". NorthShore Gut Club Quarterly Meeting. Skokie, IL.

April 20, 2018. Translational Research in Upper GI Cancers: Gastroesophageal Cancer & Cholangiocarcinoma." The University of Chicago Phase II Consortium 23rd Annual Symposium, Gleacher Center, Chicago.

Intramural

November, 2006 "Anticoagulants, Hemostasis, and Cancer – is the link c-MET? Case Presentation and Review of the literature." University of Chicago Hematology/Oncology Section Conference.

June 25, 2007 "Cancer Stem Cells". University of Chicago Hematology/Oncology Section Conference..

Oct 27, 2008 "RON Tyrosine Kinase: A Novel Molecular Target for the Treatment of Gastroesophageal Cancer." University of Chicago Hematology/Oncology Section Conference.

Sept 14, 2009 "The Role of RON Receptor Tyrosine Kinase in Gastroesophageal Cancers – Why MET is Not Enough" University of Chicago Hematology/Oncology Section Conference.

Chicago, IL, April 19, 2011. "Adjuvant Chemotherapy for Colon Cancer: Towards a Personalized Approach". University of Chicago Department of Surgery Colorectal Cancer Conference.

August 3, 2011. "Career Development Seminar for Summer Research Students". University of Chicago Laboratories.

August 29, 2011 "RON tyrosine kinase in cancer: no longer MET's Little Brother!" University of Chicago Hematology/Oncology Section Conference.

October 10, 2011. "Novel Molecularly Targeted Therapies in Esophageal Cancer: Relevance of MET&RON" Lederer Foundation Annual Meeting. University of Chicago, Chicago, IL.

Daniel Catenacci, M.D.

October 26, 2011. "Current trends in Colon Cancer Therapy: agents and approach" CANCER BIOLOGY 1: HUMAN CANCER PRESENTATION AND MODELING,

October 31, 2012 "Current trends in Colon Cancer Therapy: agents and approach" CANCER BIOLOGY 1: HUMAN CANCER PRESENTATION AND MODELING,.

November 27, 2012. "Adjuvant Chemotherapy for Colon Cancer: Towards a Personalized Approach". University of Chicago Department of Surgery Colorectal Cancer Conference. Chicago, IL,

November 14, 2012 "RON upregulation is a resistance mechanism to MET directed therapy in MET driven models." University of Chicago Department of Medicine Section of Hematology/Oncology Research Seminar, Chicago, IL.

September 25, 2013. "MET Tyrosine Kinase and GI Cancers." University of Chicago Department of Medicine Section of Hematology/Oncology AbbVie Meeting KCBD, Chicago, IL,

February 14, 2014. "Strategies to address inter- and intra- patient tumor molecular heterogeneity using next-generation companion diagnostics and PANGAEA: a novel clinical trial design". UCCCC Translational Seminars.

February 12, 2016. "Tumor molecular heterogeneity, molecular evolution, and implications in the clinic". UCCCC Translational Seminars.

March 6, 2017. Genomic Heterogeneity as a Barrier to Precision Medicine for Gastroesophageal Adenocarcinoma: An update on PANGAEA. Hematology/Oncology Section Monday Conference.

INVITED, ELECTED SERVICE:

2010-2015	University of Chicago Clinical Trials Research Committee (CTRC) member
2011-	Agency for Healthcare Research and Quality (AHRQ) case reviewer, US Department of Health and Human Services.
2012-13, 2014-17	Esophago-Gastric NCI Task Force, ALLIANCE Junior Member
2013-2015	RILOMET-1 Amgen Phase III Trial Steering Committee Member
2014-2016	University of Chicago BSD Institutional Review Board (IRB) member
2014-	Data Safety Monitoring Committee: EMD Serono anti-PDL1 phase I trial.
2014-	Cholangiocarcinoma Foundation Medical Advisory Committee Member
2015-	Debbie's Dream Foundation for Stomach Cancer Medical Advisory Committee Member
2015-	Physician Lead for the University of Chicago Cancer Center Genomic Project
2015	DOD Peer Review: Ad Hoc Reviewer Stomach Cancer
2015-2017	Hematology/Oncology Monthly Molecular Pathology Tumor Board Co-Chair
2016-	Biospecimens Committee Member
2017-	ASCO Esophageal Cancer Guideline Expert Panel Member
2018-	AACR Gastrointestinal Cancer Research Grants Scientific Review Committee – Gastric
2018-	DoD FY18 Peer Reviewed Cancer Research Program (PRCRP) – Gastric
2018	ASCO SEP 7 th edition Co-author (with B. Polite) for GI Chapter

Editorial Activities

Ad hoc Reviewer:	<i>Journal of Clinical Oncology JCO</i>	<i>Pharmacogenomics Journal</i>	<i>Tumor Biology</i>
	<i>JCO Precision Oncology JCO PO</i>	<i>Mayo Clinic Proceedings</i>	<i>JNCCN</i>
	<i>New England Journal of Medicine</i>	<i>Clinical Practice</i>	<i>Lancet Oncology</i>
	<i>Nature Reviews Disease Primers</i>	<i>Current Cancer Drug Target</i>	<i>Future Medicine</i>
	<i>Expert Review of Anticancer Therapy</i>	<i>Clinical Investigation</i>	<i>Future Oncology</i>
	<i>Journal of National Cancer Institute</i>	<i>Molecular Cancer Research</i>	<i>Cancer</i>
	<i>Inflammation & Allergy Drug Discovery</i>	<i>Trends in Molecular Medicine</i>	<i>Cancer Discovery</i>
	<i>Histology and Histopathology</i>	<i>The Oncologist</i>	<i>Colorectal Cancer</i>
	<i>World Journal of Gastroenterology (WJO)</i>	<i>Targeted Oncology</i>	<i>Oncotarget</i>
	<i>Clinical Cancer Research</i>	<i>Molecular Cancer Therapeutics</i>	<i>JAMA Oncol</i>

Associate Editor:

Daniel Catenacci, M.D.

1/2019-present *Journal of American Medical Association Network Open (JAMA Network Open)*; Oncology

Editorial Board Membership:

1/2015 –present *World Journal of Clinical Oncology (WJCO)*

7/2016 –present *Journal of Clinical Oncology (JCO) Precision Oncology*

CLINICAL PROTOCOLS:

International PI

An International Phase 1/2 Study of GRT-C901/GRT-R902, a Personalized Neoantigen Immunotherapy, in Combination with Immune Checkpoint Blockade for Patients with Advanced Solid Tumors Open to Accrual 9/2018

FIGHT: A Phase 1/3 Study of FPA144 versus Placebo in Combination with Modified FOLFOX6 in Patients with Previously Untreated Advanced Gastric and Gastroesophageal Cancer. Open to Accrual 5/2018

A Phase 1b/2 Open Label, Dose Escalation Study of Margetuximab in Combination with Pembrolizumab in Patients with Relapsed/Refractory Advanced HER2+ Gastroesophageal Junction or Gastric Cancer. Open to accrual 4/2016

A Phase II Clinical Trial of Pembrolizumab as Monotherapy and in Combination with Cisplatin+5-Fluorouracil in Subjects with Recurrent or Metastatic Gastric or Gastroesophageal Junction Adenocarcinoma (KEYNOTE 059). Open to accrual August 2015; closed to accrual 4/2016

- **Oral presentation World GI ESMO, cohort 1**
- **Author cohorts 1, 2, and 3**

A Phase III, Multicenter, Randomized, Double-Blind, Placebo Controlled Study of Rilotumumab (AMG 102) with Epirubicin, Cisplatin, and Capecitabine (ECX) as First-line Therapy in Advanced MET-Positive Gastric or Gastroesophageal Junction Adenocarcinoma. (RILOMET-1) Amgen. Open to accrual January 2013, Terminated November 2014. Closed to accrual.

- **RILOMET-1 Steering Committee Member**
- **Senior Author on final analysis abstract ASCO 2015, first author manuscript .**

National PI

NCI-MATCH – MET amplified (C1 Arm) and exon 14 deletion (C2 Arm) arms (Crizotinib) Translational Correlatives Chair. Open to enrollment 5/2016.

A Phase 1 Open-Label, Dose-Finding Study Evaluating Safety and Pharmacokinetics of FPA144 in Patients with Advanced Solid Tumors. Open to accrual January, 2016. Closed.

Investigator Initiated Trials PI:

PANGAEA -1MBBP Trial (Personalized Antibodies for GastroEsophageal Adenocarcinoma Pilot Trial) – NCT02213289. Open to accrual.

A pilot trial of perioperative mFOLFIRINOX with UGT1A1 genotyping for gastroesophageal adenocarcinoma – open to accrual 11/2014 - NCT02366819.

Understanding the Role of Genetics in Solid Tumor Malignancies. IRB 15-0443. Open to accrual 2/2016. PI: J Churpek, D Catenacci, H. Kindler. University of Chicago Medical Center.

A Multicenter Randomized Placebo-controlled Phase 2 Trial of Gemcitabine plus GDC-0449 (NSC 747691), a Hedgehog Pathway Inhibitor, in Patients with Metastatic Pancreatic Cancer (10052747). NCI/CTEP.

Opened to accrual Sept 1, 2009. (ARRA funded) Closed to accrual

Co-PI: H Kindler, D Catenacci, University of Chicago Medical Center.

Daniel Catenacci, M.D.

- Laboratory and Radiological Correlatives, D Catenacci
- Interim Analysis presented as Poster Discussion at ASCO 2012
- Final Analysis to be presented as Poster Discussion at ASCO 2013
- *Manuscript published JCO 9/2015*

2007-present: GI Tissue Banking Protocols:

- **Retrospective IRB 16146B – currently accruing**
- **Prospective Procurement IRB 16294A– currently accruing**
- **Prospective Procurement IRB XX – Internal NGS 1212 gene molecular panel**

PI: D Catenacci, University of Chicago Medical Center.

2004: Phase I/II Clinical Trial of Azacytidine and Arsenic Trioxide combination treatment of Myelodysplastic Syndromes.

PI: G Schiller, UCLA Medical Center. *Closed to accrual*

Site PI Pharma Sponsored:

A Phase 3, Global, Multi-Center, Double-Blind, Randomized, Efficacy Study of IMAB362 Plus mFOLFOX6 Compared with Placebo Plus mFOLFOX6 as First-line Treatment of Subjects with Claudin (CLDN)18.2-Positive, HER2-Negative, Locally Advanced Unresectable or Metastatic Gastric or Gastroesophageal Junction (GEJ) Adenocarcinoma. *Open to Accrual 2/1/19.*

Trial Steering Committee Member.

A Phase III, Randomized, Double-Blind, Clinical Trial of Pembrolizumab (MK-3475) plus Chemotherapy (XP or FP) versus Placebo plus Chemotherapy (XP or FP) as Neoadjuvant/Adjuvant Treatment for Subjects with Gastric and Gastroesophageal Junction (GEJ) Adenocarcinoma (KEYNOTE-585). *Accrual on hold until FLOT-pembro phase I completed.*

An International Phase 1/2 Study of GRT-C901/GRT-R902, a Personalized Neoantigen Immunotherapy, in Combination with Immune Checkpoint Blockade for Patients with Advanced Solid Tumors *Open to Accrual 9/2018*

A Phase 1/3 Study of FPA144 versus Placebo in Combination with Modified FOLFOX6 in Patients with Previously Untreated Advanced Gastric and Gastroesophageal Cancer. *Open to Accrual 5/2018*

A Phase 1/2, Open-Label, Safety, Tolerability, and Efficacy Study of Epacadostat in Combination with Pembrolizumab and Chemotherapy in Subjects with Advanced or Metastatic Solid Tumors (ECHO-207/KEYNOTE-723). *Open to accrual 11/2017*

Combination with Cisplatin and 5-Fluorouracil versus Placebo in Combination with Cisplatin and 5-Fluorouracil as First-Line Treatment in Subjects with Advanced/Metastatic Esophageal Carcinoma (KEYNOTE-590). *Open to accrual 10/2017*

A Phase III, Multicenter, Randomized, Double-Blind, Placebo-Controlled Study of AG-120 in Previously-Treated Subjects with Nonresectable or Metastatic Cholangiocarcinoma with an IDH1 Mutation *Open to accrual May 2017*

A Randomized, Multicenter, Double Blind, Phase III Study of Nivolumab or Placebo, in Subjects with Resected Lower Esophageal, or Gastroesophageal Junction Cancer. *Open to accrual 8/2016.*

A Phase 1b/2 Open Label, Dose Escalation Study of Margetuximab in Combination with Pembrolizumab in Patients with Relapsed/Refractory Advanced HER2+ Gastroesophageal Junction or Gastric Cancer. *Open 4/2016.*

A Phase II, Open-Label, Single-Arm, Multicenter Study to Evaluate the Efficacy and Safety of INCB054828 in Subjects with Advanced/Metastatic or Surgically Unresectable Cholangiocarcinoma Including FGFR2 Translocations who Failed Previous Therapy Closed to accrual 10/2018

Daniel Catenacci, M.D.

A Phase 1b/2 Study of MEDI4736 in Combination with Tremelimumab, MEDI4736 Monotherapy, and Tremelimumab Monotherapy in Subjects with Metastatic or Recurrent Gastric or Gastroesophageal Junction Adenocarcinoma. Closed to accrual 5/2018

A Phase 1b Open-Label Study of PEGylated Recombinant Human Hyaluronidase (PEGPH20) Combined with Pembrolizumab in Subjects with Selected Hyaluronan-High Solid Tumors. Closed to accrual 4/2018.

A Phase 2, Open-Label, Randomized Study to Evaluate the Efficacy and Safety of GS-5745 Combined with Nivolumab versus Nivolumab Alone in Subjects with Unresectable or Recurrent Gastric or Gastroesophageal Junction Adenocarcinoma. Closed to accrual 4/2017

Randomized, Double-Blind Phase 3 Study Evaluating TAS-102 Plus Best Supportive Care (BSC) Versus Placebo Plus BSC in Patients with Metastatic Gastric Cancer Refractory to Standard Treatments. Open to Accrual 4/2016, closed to accrual 11/30/17

A Phase 2, Open-label Evaluation of CRS-207 and Pembrolizumab in Adults with Recurrent or Metastatic Gastric, Gastroesophageal Junction, or Esophageal Adenocarcinomas. Terminated early.

A Randomized, Active-Controlled, Partially Blinded, Biomarker Select, Phase III Clinical Trial of Pembrolizumab as Monotherapy and in Combination with Cisplatin+5-Fluorouracil versus Placebo+Cisplatin+5-Fluorouracil as First-Line Treatment in Subjects with Advanced Gastric or Gastroesophageal Junction (GEJ) Adenocarcinoma (KEYNOTE 062). Open to Accrual 3/2016 Closed to Accrual 6/2017.

A021302: Impact of Early FDG-PET Directed Intervention on Preoperative Therapy for Locally Advanced Gastric Cancer: A Random Assignment Phase II Study. Open to accrual October 2015.

A Phase 1 Open-Label, Dose-Finding Study Evaluating Safety and Pharmacokinetics of FPA144 in Patients with Advanced Solid Tumors. Open to accrual January, 2016. Closed.

A Phase II Clinical Trial of Pembrolizumab as Monotherapy and in Combination with Cisplatin+5-Fluorouracil in Subjects with Recurrent or Metastatic Gastric or Gastroesophageal Junction Adenocarcinoma (KEYNOTE 059). Open to accrual August 2015; closed to accrual 4/2016

ARQ197-A-U303 A Phase III, Randomized, Double-Blind Study of Tivantinib (ARQ 197) in Subjects with MET Diagnostic-High Inoperable Hepatocellular Carcinoma (HCC) Treated with One Prior Systemic Therapy. Open to accrual January 2013. Closed to accrual.

A Double-Blind, Placebo-Controlled, Randomized, Multicenter Phase III Study Evaluating the Efficacy and Saftery of Pertuzumab in Combination with Trastuzumab and Chemotherapy in Patients with Her2-Positive Metastatic Gastroesophageal Junction and Gastric Cancer (JACOB study). Open to accrual July 2013. Closed to accrual.

A phase I open-label, non-randomized, dose-escalation first-in-man trial to investigate the c-Met kinase inhibitor EMD 1214063 under two different regimens in subjects with advanced solid tumors. Phase I expansion for MET amplification. Open to accrual November, 2013. Closed to accrual.

Phase 1b Multi-Cohort Study of MK-3475 in Subjects with Advanced Solid Tumors - Gastric/EGJ cohort PI for anti-PD1 inhibitor. Open to accrual November, 2013. Closed to accrual.

Phase 1, First-in-Human Study Evaluating the Safety, Tolerability, and Pharmacokinetics of AMG 337 in Adult Subjects with Advanced Solid Tumors. Open to accrual September 2013. Closed to accrual.

A Phase III, Multicenter, Randomized, Double-Blind, Placebo Controlled Study of Rilotumumab (AMG 102) with Epirubicin, Cisplatin, and Capecitabine (ECX) as First-line Therapy in Advanced MET-Positive Gastric or Gastroesophageal Junction Adenocarcinoma. (RILOMET-1) Amgen. Open to accrual January 2013, Terminated November 2014. Closed to accrual.

Daniel Catenacci, M.D.

- *RILOMET-1 Steering Committee Member*
- **Senior Author on efficacy abstract ASCO 2015**

A Randomized, Phase III, Multicenter, Double-Blind, Placebo-Controlled Study Evaluating The Efficacy And Safety Of Onartuzumab (Metmab) In Combination With 5-Fluorouracil, Folinic Acid, And Oxaliplatin (mFOLFOX6) In Patients With Metastatic HER2-Negative, Met-Positive Gastroesophageal Cancer. Open to accrual July 2013. Closed to accrual.

A Pilot Study of neoadjuvant and adjuvant mFOLFIRINOX in localized, resectable pancreatic adenocarcinoma. Co-PI (Kindler). Closed to accrual.

A Phase 2b Randomized, Open-Label Trial of JX-594 (Vaccinia GM-CSF / TK-deactivated Virus) Plus Best Supportive Care Versus Best Supportive Care in Patients with Advanced Hepatocellular Carcinoma Who Have Failed Sorafenib Treatment. Jennerex. Closed to accrual.

ECOG E1208: A Phase III Randomized Trial of Chemoembolization with or without Sorafenib in Unresectable Hepatocellular Carcinoma (HCC) in Patients with and without Vascular Invasion. Closed to accrual.

CALGB 80802: Phase III randomized study of sorafenib (IND 69896, NSC 724772) plus doxorubicin versus sorafenib in patients with advanced hepatocellular carcinoma (HCC). Closed to accrual.

A Phase 1, Open-Label, Dose Escalation Study of ASG-5ME in Patients with Pancreatic or Gastric Adenocarcinoma. Seattle Genetics. Closed to accrual.

A Randomized, Phase II, Multicenter, Double-Blind, Placebo-Controlled Study Evaluating The Efficacy and Safety of Onartuzumab (MetMab) in Combination with 5-Fluorouracil, Folinic Acid, and Oxaliplatin (mFOLFOX6) in Patients with Metastatic HER2-Negative Gastro-esophageal Cancer. Genentech. Closed to Accrual.

Randomized, Double Blind, Phase II Study of FOLFOX Bevacizumab with MetMab versus Placebo as First Line Treatment for Patients with Metastatic Colorectal Cancer. Genentech/Sarah Cannon. Closed to Accrual.

SWOG S0809: A Phase II Trial of Adjuvant Capecitabine/Gemcitabine Chemotherapy Followed by Concurrent Capecitabine and Radiotherapy in Extrahepatic Cholangiocarcinoma (EHCC). SWOG. Closed to Accrual.

A Multicenter Randomized Placebo-controlled Phase 2 Trial of Gemcitabine plus GDC-0449 (NSC 747691), a Hedgehog Pathway Inhibitor, in Patients with Metastatic Pancreatic Cancer (10052747). NCI/CTEP. Opened to accrual Sept 1, 2009. NCI/CTEP. Closed to accrual (ARRA funded)

A Randomized, Double Blind Placebo Controlled Phase 2 Study of FOLFOX plus or minus GDC-0449 in patients with advanced gastric and gastroesophageal junction (GEJ) carcinoma. NCI/CTEP. Closed to accrual.

A Multicenter Random Assignment Phase II Study of Irinotecan and Alvocidib (flavopiridol) versus Irinotecan Alone for Patients with p53 wild type Gastric Adenocarcinoma NCI/CTEP. Closed to accrual.

A Phase 2, Randomized, Double-blind, Placebo-controlled Study Evaluating the Safety and Efficacy of FOLFIRI in Combination With AMG 479 or AMG 655 Versus FOLFIRI for the Second-line Treatment of KRAS-mutant Metastatic Colorectal Carcinoma. Amgen. Closed to accrual.

Daniel Catenacci, M.D.

TEACHING ACTIVITIES:

University of Chicago Medical Center, Chicago, Illinois. 2010-present

For the College (B.A., B.S.):

- (a) Didactic
 - 2011- Annual Lecture on Career Development Seminar for Summer Research Students
- (b) Clinical
 - 2011- Preceptor weekly for undergraduate students (1 student per year).

For Graduate Programs (Masters, Ph.D.):

- (a) Didactic
 - 2011- Graduate Course CANCER BIOLOGY I CABI 30800: HUMAN CANCER PRESENTATION AND MODELING: Annually one lecture on Colon Cancer
 - Graduate Course CANCER BIOLOGY III
 - Annually one lecture on Gastroesophageal Cancer
 - 2011- Annual Lecture on Career Development Seminar for Summer Research Students

For Pritzker School of Medicine (M.D.):

- (a) Didactic
 - 2011- Career Development In Oncology presentation one lecture per inpatient rotation.
 - 2011- Annual Lecture on Career Development Seminar for Summer Research Students
 - 2014 Teaching Assistant for MEDC-30011: Epidemiology and Research Design
 - Epidemiology and Research Design MEDC-30011 Medical Student (year 1) Course: Small group session Instructor.
- (b) Clinical
 - 2010- Daily inpatient rounding 4 weeks per year. 0-2 students per rotation.
 - 2011- M1 Longitudinal Program Preceptor weekly (1-2 students).

For Graduate Medical Education (Residency and Clinical Fellowships):

- (a) Didactic
 - 2010- Discussant, Medical Oncology Fellows Journal Club, Grant Writing, Board Review.
 - 2010- Annual Lecture to First Year Fellows on "Gastroesophageal", "Cholangiocarcinoma", and "Translational Medicine Basics".
 - 2012 Discussant, Internal Medicine Residents Clinical-Pathologic Correlates Conference
 - 2015 "Meeting Highlights: Gastrointestinal Cancer." ASCO Trainee & Early-Career Oncologist Lounge. ASCO 2015, Chicago IL.
 - 2016- Annual Lectures to Surgical Residents and Fellows (General and Cardiothoracic) for Esophagogastric Cancer.
- (b) Clinical
 - 2010- Daily inpatient rounding 4 weeks per year ~2-4 residents/interns, 2 fellows per rotation.
 - 2010- Supervision of fellows, residents in outpatient GI oncology clinic weekly (0-3 fellows)

University of Chicago Medical Center, Chicago, Illinois. 2006-2010:

- Medical Student Lecture Series
 - "Myelodysplasia", "Mesothelioma", "Hepatocellular Carcinoma".

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UCLA Medical Center, Westwood, California. 2004-2006:

- Distinguished Teacher Award for UCLA Interns and Medical Students.
 - Teaching clinical medicine to Interns and Medical Students.

Wayne State University Medical School, Detroit, Michigan. 2000-2003:

- Tutor for individual and group sessions:
 - Anatomy, Histology, Biochemistry, Physiology, Neuroscience.

University of Waterloo, Waterloo, Ontario, Canada 1995-1999:

- Teacher Assistant in Science laboratories at University of Waterloo:
 - Cell Biology, Histology, Microbiology, 1997-1999

Lambton Kent District School Board, Sarnia, Ontario, Canada. 1997-1999:

- Summer School Teacher Assistant
 - Mathematics, Grades 9-12.

University of Chicago Research Trainees/Mentees:

Highschool Mentorship:

2012: IMSA (Igniting and Nurturing Creative, Ethical Scientific Minds that advance the human condition

Jiwon Kwak

Nitya Pariti

2018: Ocean Malka Chicago EYES on Cancer Summer Study

Undergraduate Mentorship

2011: Ciara Zagaja – Laboratory Fellowship June-August 2011.

Graduate School Mentorship

2015/16: Sravya Tumuluru - preparation for Graduate School at University of Chicago Medicine & Biological Sciences, as a technician in my laboratory

Tumuluru S, Xu D, Xu P, Henderson L, Catenacci DVT. Targeted therapies for targeted populations: MET inhibition for *MET* amplified gastroesophageal cancer. *In Preparation*

Medical Student Letters/Mentorship/Teaching

2009- : Mohamed El Dinali – clinical reference letter

John Wojcik - clinical reference letter

Obinna Orji – clinical reference letter

Longitudinal Program at Pritzker for MS1

2010-2011: Christine Anterasian

David Bluhm

2011-2012: Claire Naus

Alan Hutchison

2012-2013: Chenyu Lin

Arjun Dayal

2013-2014 Jennifer Jones (MS3)

Guarav Ajmani

Daniel Camacho

2014-2015 Chijioke "CJ" Ikente

2015-2016 Chantai Tian

2011 University of Chicago Pritzker Summer Research Program – Cluster Group Leader

Resident Mentorship

2010: Anna Halpern – (yr 3) clinical reference letter, career development

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2014: Andrew Hantel – (yr 1) translational research in GI malignancies
 2017-2018 Syed Abdur-Rahman – clinical trial coordinator, internal medicine residency letter application

Fellow Mentorship

- 2010: Manish Sharma - OSI-906 clinical LOI/protocol design and submission
 2011: Ahad Sadiq - ARQ197 clinical LOI for GEC first line metastatic.
 Dan Geynisman - ARQ197 clinical LOI/correlates papillary renal cancer, Cholangiocarcinoma
 2012: Emilio Araujo Mino - clinical reference letter
 Dan Geynisman - Upper GI Malignancies
 2013: Amikar Sehdev - Upper GI Malignancies
 Erica Ramsdale - Upper GI Malignancies
 Vassiliki Saloura - Upper GI Malignancies
 Andrea Amico - Family Risk of Upper and other GI Malignancies
 2014: Andrea Amico - Family Risk of Upper and other GI Malignancies
- Amico A, Nielsen S, Geynisman D, Rambo B, Carey GB, Gulden C, Facekenthal J, Olopade O, **Catenacci D**. Challenges of applying tumor genome analysis to the germline: Examples from GI Oncology. AACR Cancer Susceptibility and Cancer Susceptibility Syndromes conference. San Diego, CA. January 29-February 1, 2014.
- Hollis Walker - Upper and other GI Malignancies
 Jen Veneris - Upper GI Malignancies
 Steve Maron - GI database procurement/Colon cancer expression analysis mass spec
 Chris Chapman - Upper GI malignancies, EUS portal venous sampling for CTCs and cfDNA
- Waxman I, Chapman C, Koons A, Konda V, Siddiqui U, Gelrud A, Xu P, **Catenacci DVT**. Detection of Portal Vein (PV) Circulating Tumor Cells (CTCs) in Pancreatic Cancer (PC) patients obtained by EUS guided PV Sampling. A safety and Feasibility trial. Accepted as an Oral Presentation. Digestive Disease Week 2014. Chicago, IL. May 4-6, 2014.
- 2015/16: Steve Maron -Immuno-Oncology Molecular Profiling of esophagogastric cancer
 -GI database procurement/Brain metastases project
- Maron S, Luke J, Hovey R, Bao R, Gajweski T, Ji Y, Seiwert T, **Catenacci DVT**. Molecular Characterization of T-Cell-Inflamed Gastroesophageal Cancer. WIN 2016, Paris France. Best Abstract and Oral Presentation.
- Hollis Walker - Upper and other GI Malignancies
 Nanna Sulai - Upper and other GI Malignancies
 Shuang Q Zhang - Pancreatic Cancer and other Upper and other GI Malignancies
- Zhang SQ, **Catenacci DVT**. How can next-generation diagnostics aid pancreatic adenocarcinoma treatment? *Future Oncology* Mar 2016. 26831761
- 2016/17: Steve Maron -Immuno-Oncology Molecular Profiling of esophagogastric cancer
 -GI database procurement/Brain metastases project
- Maron S, Luke J, Hovey R, Bao R, Gajweski T, Ji Y, **Catenacci DVT**. SITC annual conference, Orlando FL Feb, 2017. Identification of T cell-inflamed gastric adenocarcinoma in TCGA
 - *Pectasides E, *Stachler MD, *Dersk S, *Lui Y, *Maron S, Islam M, Alpert L, Kwak H, Kindler HL, Polite BP, Sharma MR, Allen K, O'Day E, Lomnicki S, Maranto M, Kanteti R, Fitzpatrick C, Weber C, Setia N, Xiao SY, Hart J, Nagy RJ, Kim KM, Choi MG, Min BH, Nason KS, O'Keefe L, Watanabe M, Baba H, Lanman R, Agonston T, Oh DJ, Dunford A, Thorne AR, Ducar MD, Wollison BM, Coleman HA, Ji Y, Posner M, Roggin K, Turaga K, Chang P, Hogarth K, Siddiqui U, Gelrud A, Ha G, Freeman SS, Rhoades J, Reed S, Gydush G, Rotem D, Davison J, Imamura Y, Adalsteinsson, Lee J, Bass AJ, **Catenacci DVT**. Genomic Heterogeneity as a Barrier to Precision Medicine in Gastroesophageal Adenocarcinoma. *Epub ahead of print Oct 4 Cancer Discovery* 2017. PMID 28978556 * co-first authors
 - Maron SB, **Catenacci DVT**. Novel Targeted Therapies for Esophagogastric Cancer. *Epub Surg Oncol Clin N Am* 2017. PMID: 28279470.
 - Maron SB, **Catenacci DVT**. Update on Gastroesophageal Adenocarcinoma Targeted Therapies. *Epub Hematol Oncol Clin North Am* 2017. PMID: 28501091
 - Maron SB, Lomnicki S, Chase L, Joshi S, Nagy B, Lanman R, Lee J, Catenacci DVT. 'Genomic

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landscape of cell-free DNA in patients with gastroesophageal adenocarcinoma' *manuscript in preparation*.

- Sope Olugbile - TCR sequencing in patients (MSI-H) receiving immunotherapies
- Kevin Wood - Upper and other GI Malignancies
- Smita Joshi - Merck LOI perioperative immunotherapy for gastroesophageal cancer
- Joshi SS, Maron SB, Catenacci DVT. Pembrolizumab for treatment of advanced gastric and gastroesophageal junction adenocarcinoma. Nov 2. doi: 10.2217/fon-2017-0436. [Epub ahead of print] 2017 *Future Oncology*. PMID: 29094609

2017/19: Smita Joshi – GI clinical oncology, clinical trials, translational GI research

Anu Neerukonda – GI clinical oncology

2018- Natalie Reizer – GI Oncology, Pharmacogenomics Program, UGT1A1 genotype directed dosing of irinotecan in GI malignancies.

- Clinical trial IIT: A Phase 1 Dose Titration Study of UGT1A1 genotype directed dosing of Irinotecan combined with 5FU, leucovorin, oxaliplatin and docetaxel in patients with advanced upper gastrointestinal adenocarcinoma

Catenacci Lab Castle Foundation Scholars and Mentorship

The Castle Foundation Award is a gift provided by the Castle Foundation with intention for the fostering and training of students in the research of gastroesophageal cancer and other gastrointestinal malignancies. The funding is to support research conducted, with my supervision and mentorship, with the participation of i) students who have completed their Undergraduate Degree with the intention of applying to various Graduate Schools in in the medical field (Medical School, Graduate School, Nursing School, Physician Assistant School) or ii) Fellows completing their Oncology Fellowship with intention to have a career in Academia. A recent gift of \$1.475million over 5 years was given to this translational/laboratory research effort.

2013-2016: Les Henderson – career development --> Senior Cytogenetic Technologist, WI

- Henderson L, Peng Xu, Rambo B, Liao WL, J, Hembrough T, Catenacci DVT. KRAS gene amplification defines a distinct molecular subgroup of gastroesophageal adenocarcinoma that may benefit from combined anti-RAS/RAF/MEK/ERK and PIK3/PTEN/mTOR/AKT pathway inhibition. AACR KRAS Feb 21-24, 2014. Orlando FL (Abstr 55).

2013-14: Brittany Rambo Physician Assistant (see contributions in publication list above)

2015-16: Rachel Rendak Nurse Practitioner (see contributions in publication list above)

2014-16: Emily O'Day Physician Assistant (see contributions in publication list above)

2016-18: Samantha Lomnicki Medical School (see contributions in publication list above)

2017-2019 Steve Maron Coggeshall Fellow/Castle Foundation Scholar → MSKCC 12/01/18

K12 2018 - "The impact of intra-patient tumor genomic heterogeneity on immune environment heterogeneity and immune checkpoint blockade resistance."

ASCO YIA 2018 "Intra-patient tumor immune environment heterogeneity and immune checkpoint blockade resistance."

AACR YIA 2018 "Intra-patient tumor heterogeneity and checkpoint blockade resistance."

- Maron S, Alpert L, Kwak HA, Lomnicki S, Chase L, Xu D, O'Day E, Nagy RJ, Lanman RB, Cecchi F, Hembrough T, Hart J, Xiao SY, Setia N, Catenacci DVT. Targeted therapies for targeted populations: anti-EGFR therapy for EGFR amplified gastroesophageal adenocarcinoma. *Epub ahead of print Feb 15 Cancer Discovery* 2018. PMID 29449271

2017-2019 Leah Chase Medical School Candidate (see contributions in publication list above)

2019- Nicole Arndt

Natalie Reizine – Pharmacogenomics and Heme/Onc Fellowship

Development of UGT1A1 genotyping and dose finding study with irinotecan in advanced GI malignancies.

Journal of Clinical Oncology Precision Oncology/ASCO Reviewer Mentorship Program:

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2019-2020 Lorenzo Gerratana, Xuemei Ji, Andrea Napolitano.

1. 8/4/20 Young vs Makhdoom – For Defense. Colorectal cancer.
2. 2/24/20 and 8/20/19 Creech vs CAROLINA RADIOLOGY CONSULTANTS, P.A. and JEFFREY E. JONES, M.D. For Plaintiff. Hepatocellular cancer.
3. 10/22/19 Gross vs BNSF. For Plaintiff. Asbestos and colorectal cancer.
4. 8/2/19 Lopez vs United States. For Defense. Colorectal cancer.
5. 2/24/19 Benjamin Torres vs William Summers MD For Plaintiff. Anal Cancer.
6. 1/14/19 Allen v. St. Luke's, et al. For Plaintiff. Esophagogastric MANEC tumor (mixed adeno and neuroendocrine cancer) .
7. 5/11/18 Teresa Brown vs. Jatinder S. Sekhon, M.D. For Defense. Colorectal cancer.
8. 6/7/17 Larry Ames v. Donne Graessle D.O. For Plaintiff. Colorectal Cancer.
9. 2/24/17 Lerner vs Kelly. For Plaintiff. Colorectal Cancer.
10. 1/25/17 Robert Adams v. Poirot & Macoupon Family. For Defense. Small Bowel Carcinoid Tumor.